Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Chapter 4

Diseases of the Gastrointestinal System

CHRISTINE B. NAVARRE AND D.G. PUGH

The gastrointestinal system is, arguably, more prone to disease than any other part of the sheep or goat. Gastrointestinal parasitism alone is the most significant cause of production and animal losses in much of North America.[1,2] There is no substitute for a thorough physical examination when trying to determine the affected body systems of a sick animal; this is especially true in diseases of the gastrointestinal system. A complete physical examination should include palpation for body condition, assessment of abdominal shape and rumen motility, observation of the consistency of the stool, and evaluation for the presence of bloat. However, because rectal palpation cannot be performed in sheep and goats, diagnosis of disease in a particular segment of the gastrointestinal system can be difficult. Therefore, the clinician may have to perform ancillary diagnostic procedures to characterize gastrointestinal diseases properly.

DIAGNOSTIC PROCEDURES

Clinicopathologic Data

Clinicopathologic data consisting of a complete blood count (CBC), serum biochemical evaluation (SBE), and urinalysis can be helpful in differentiating gastrointestinal diseases, developing a prognosis and plan for treatment, and monitoring treatment. A CBC rarely identifies a specific disease, but it can be helpful in evaluating the severity of dehydration, anemia, and hypoproteinemia. The clinician must take care to interpret the packed cell volume (PCV) and total protein in light of the hydration status of the animal as noted on physical examination. An anemic or dehydrated hypoproteinemic animal may have normal PCV and total protein values. Both the CBC and SBE can be helpful in characterizing the presence and severity of an inflammatory disease process. Changes in the total and differential white blood cell count indicate acute or chronic inflammation; increases in globulins or fibrinogen suggest a chronic inflammatory disease. Low protein levels, especially albumin, can point to chronic blood loss from intestinal parasitism or infiltrative bowel disease. Liver disease should be suspected if liver enzymes or bilirubin are elevated. However, liver enzymes can be normal in chronic liver disease. Also, albumin levels rarely drop in ruminants with liver disease, as they do in other species.[3] Changes in electrolytes can occur with gastrointestinal disease, especially if the animals are anorexic. Electrolyte measurements also are helpful in formulating a treatment plan. Hypochloremia and metabolic alkalosis occasionally occur in abomasal disease. A mild hypocalcemia may be encountered in some small ruminants with gastrointestinal atony. Because many animals with gastrointestinal disease are dehydrated and therefore azotemic and possibly hypoproteinemic, urinalysis is helpful to eliminate urinary disease as a cause of these pathologies.

Normal ranges for clinicopathologic values are included in this textbook (see Appendix III) and also have been published in several other textbooks.[4-7] However, clinicians would do well to learn the normal values, especially serum biochemistry values, established by the laboratory most commonly used for analysis.[7]

Rumen Fluid Analysis

Analysis of rumen fluid can help differentiate diseases of the forestomachs. An appropriately sized orogastric tube can be passed through the oral cavity for fluid collection (Figure 4-1). The clinician must properly restrain the animal, using a mouth speculum (Figure 4-2) to prevent tube chewing. If the tube is chewed, its roughened surface may damage the esophagus; parts of a broken tube can be swallowed. Rumen fluid also can be collected using percutaneous rumenocentesis[4,8-12] (Figure 4-3). A 16-gauge

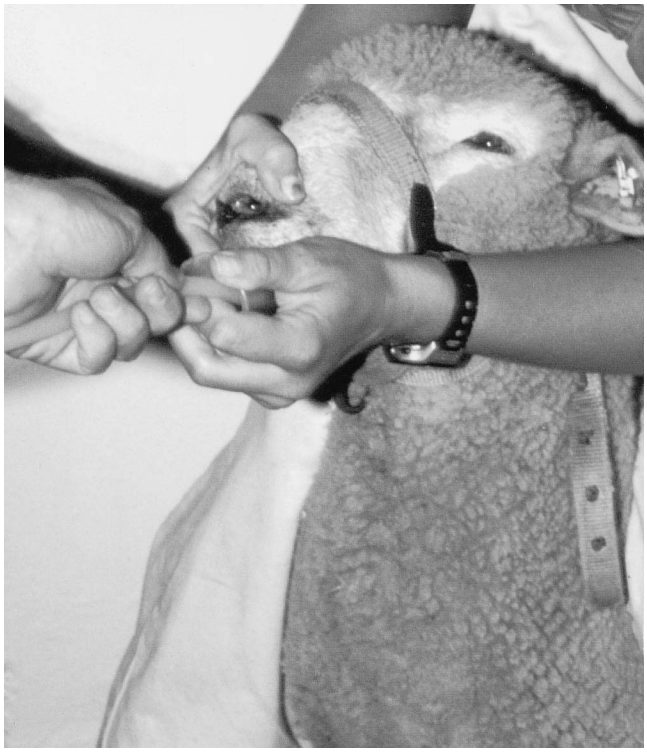

Figure 4-1 Passage of an orogastric tube through a mouth speculum. The tube should be lubricated and passed slowly down the esophagus.

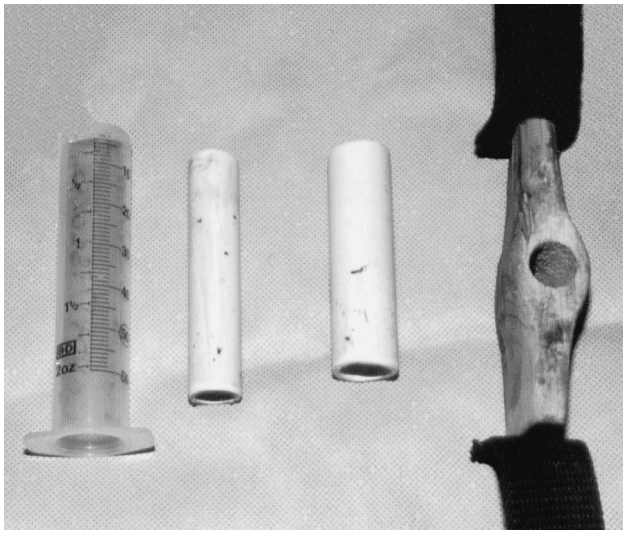

Figure 4-2 Mouth speculums for use in passing an orogastric tube. Various equipment designs can be used to protect the tube from being chewed.

needle can be inserted in the rumen through the abdominal wall caudal to the xyphoid and to the left of midline. The clinician then aspirates fluid with a syringe. Local anesthesia and sedation of the animal may be necessary. This technique avoids the saliva contamination that can occur during collection with an orogastric tube, and it appears to be less stressful. Rumenocentesis presents a slight risk of peritonitis, but this risk can be minimized with proper restraint. Percutaneous rumenocentesis should not be performed on pregnant females.

Figure 4-3 The site for performing a rumenocentesis. The area should be clipped, cleaned, and surgically prepared.

After the fluid is collected, it can be analyzed for color, odor, pH, protozoal species and motility, methylene blue reduction time (MBR), Gram's staining characteristics, and chloride levels. Normal values are listed in Table 4-1. Anorexia may cause the fluid to appear darker, the pH to increase, and the number and motility of protozoa to decrease. A gray color, low pH, and dead or no protozoa are seen in rumen acidosis from grain overload. The MBR is prolonged with any type of indigestion. Large numbers of gram-positive rods (*Lactobacillus* species) also may be seen in rumen acidosis. Elevated rumen chloride indicates an abomasal or proximal small intestinal obstruction (either functional or mechanical).

Fecal Examination

The most important reason for examining feces in sheep and goats is to determine the presence and relative number of nematode parasites infesting an animal or flock. The quantitative technique for determining eggs per gram of feces (EPG) is shown in Box 4-1. Fecal EPG values of more than 500 to 1000 indicate serious infestation and the need for intervention.

Fecal occult blood testing and acid-fast staining of fecal smears also can be performed. Fecal occult blood tests can detect microscopic amounts of blood in the feces. However, they cannot indicate which part of the gastrointestinal tract is bleeding. Acid-fast stains of fecal smears that reveal clumps of acid-fast rods usually indicate infection with *Mycobacterium paratuberculosis* (Johne's disease). Generally, individual acid-fast rods found on fecal examination are nonpathogenic.

TABLE 4-1

NORMAL RUMEN FLUID CHARACTERISTICS OF SHEEP AND GOATS

CHARACTERISTIC	NORMAL VALUES
Color	Green
Odor	Aromatic
pH*	6.5 to 7.5
Protozoa†	Mixed sizes and species rapidly moving
Methylene blue reduction time‡	3 to 6 minutes
Gram's stain	Gram-negative rods predominate
Rumen chloride	Less than 25 to 30 mEq/L

From Nordlund KV, Garrett EF: Rumenocentesis: a technique for collecting rumen fluid for diagnosis of subacute rumen acidosis in dairy herds, *Bovine Pract* 28:109, 1994; Keefe GP, Ogilvie TH: Comparison of oro-ruminal probe and rumenocentesis for prediction of rumen pH in dairy cattle, *Proc 30th Ann Am Assoc Bovine Pract Conv,* p 168, 1997; Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
*Use pH paper with at least 0.5-unit gradations.
†Place a drop of fluid on a warm slide and cover with a coverslip. Examine under 100× magnification.
‡Mix one part 0.03% methylene blue to 20 parts rumen fluid. Measure time for blue color to clear to match a control tube of fluid.

Abdominocentesis

Abdominocentesis is useful in discerning the causes of fluid distention in the abdomen. Two methods can be used. The first technique involves tapping the lowest point of the abdomen slightly to the right of midline; it is useful in ruling out a ruptured bladder as the cause of general ascites (Figure 4-4).[4,13] The clinician should take care to avoid the prepuce in males. The second technique is useful if peritonitis is suspected. Because localized peritonitis is more common than generalized peritonitis, four sites are tapped.[14] The two cranial sites are slightly caudal to the xyphoid and medial to the milk veins on the left and right sides. The two caudal sites are slightly cranial to the mammary gland and to the left and right of midline. For either technique, manual restraint with sedation is recommended; the use of real-time ultrasonography may help locate fluid pockets. A 20-gauge needle or teat cannula can be used for fluid collection.[13] The clinician should prepare the site using sterile technique and provide local anesthesia when employing a teat cannula. Fluid should be collected in a small ethylenediamine tetra-acetic acid (EDTA) tube for analysis and a sterile tube for culture. Abdominal fluid can be difficult to obtain because of the small amounts normally present in both sheep and goats. The clinician should minimize the ratio of EDTA to fluid because EDTA can falsely elevate protein levels. Using EDTA tubes made for small animals or shaking excess EDTA out of large tubes resolves this problem. Normal culture values are similar to those for cattle (clear, colorless to slightly yellow, 1 to 5 g/dl protein, less than 10,000 cells).[14] Cytologic examination

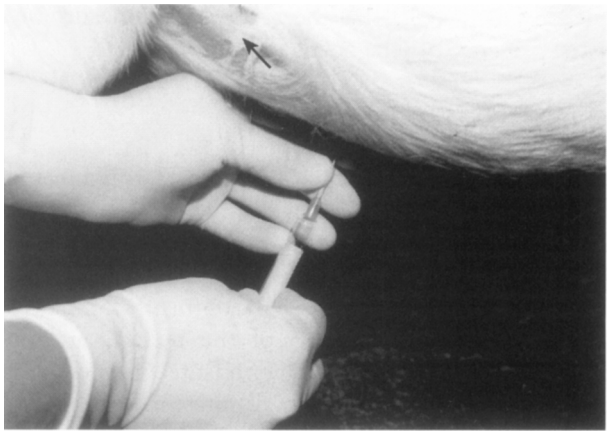

Figure 4-4 Ventral and caudal sites for performing abdominocentesis. The needle indicates the ventral site. The caudal site is the clipped area below the flank *(arrow).*

BOX 4-1

McMaster's Quantitative Technique for Obtaining the Number of Nematode Eggs Per Gram of Feces

1. Weigh 2 g of feces and thoroughly mix with 28 ml of water. This is the preferred method. However, if a gram scale is not available, feces can be added to the 28 ml of water until the water level indicates 30 ml. This approximates 2 g of feces.
2. Remove 1 ml of well-mixed fecal-water suspension, add to 1 ml of Sheather's solution,* and mix well.
3. Fill both sides of a McMaster's chamber with the Sheather's solution–fecal-water mixture.
4. Allow to stand for 5 minutes.
5. Count ova inside of chamber lines on both sides. Multiply the number of ova by 100. This number approximates the number of eggs per gram of feces.

*Sheather's solution consists of 1470 ml distilled water, 5 lb sugar, and 30 ml liquid phenol. It is made by heating distilled water and sugar in the upper half of a double boiler until the sugar is dissolved, cooling, and then adding the phenol.

is needed to characterize the cell population and assess for the presence of phagocytized bacteria.

Radiography

Radiography of the abdomen can be performed in small ruminants using small animal techniques. In adults, the rumen normally fills the entire abdomen. Radiography can detect gas distention of the small intestine, abdominal fluid, and foreign bodies.[14,15] Contrast techniques are useful for diagnosing atresia of the rectum or colon. Unlike in other small animals, contrast techniques are not practical for characterizing small intestinal problems in sheep and goats because the rumen dilutes and slows passage of the contrast media.[16]

Ultrasonography

Ultrasonography can be used to provide better characterization of abdominal distention, internal and external abdominal masses, and gross lesions of the liver. Ascites may be differentiated from fluid in the intestinal tract, and gas distention of the intestines can be differentiated from fluid distention. Normal ultrasonographic examination of the liver in sheep has been described.[17] The liver can be viewed on the right side from the seventh or eighth rib caudally to the thirteenth rib (Figures 4-5 and 4-6). Ultrasonography can be used to perform biopsies of organs or masses and to locate pockets of fluid.

Laparoscopy

Laparoscopy is more commonly used as a reproductive tool, but it also can be used diagnostically as an alternative to exploratory laparotomy in small ruminants.[18,19] General anesthesia is recommended. The technique for laparoscopic exploration of the abdomen used for cattle can be modified for use in sheep and goats.[20] The clinician inserts a cannula in the caudal abdomen and carefully inflates the abdomen with carbon dioxide (CO_2). With the animal restrained in dorsal recumbency and either sedated or anesthetized, the clinician places the cannula in the inguinal area as described for laparoscopic insemination in Chapter 6. Entrance on the right side allows visualization of most of the abdominal organs. The clinician should avoid the rumen when introducing the laparoscope into the abdomen. This procedure may be enhanced by lowering the head or rear of the animal, allowing better visualization of the entire abdomen. Animals should be properly ventilated during this procedure because inflation of the abdomen and lowering of the head can put pressure on the diaphragm.

Exploratory Laparotomy

Exploratory laparotomy can be a valuable diagnostic tool in evaluating gastrointestinal diseases when other tests indicate abdominal disease. In some cases, therapeutic surgical techniques can be performed at the same time. The technique of exploratory laparotomy used in cattle can be adopted for sheep and goats as long as the clinician keeps in mind that these animals are more likely to lie down during surgery and standing surgery should only rarely be attempted.[21] Small ruminants should be heavily sedated or placed under general anesthesia during this procedure. They may show signs of postoperative pain, anorexia, and depression and should be treated accordingly with a nonsteroidal antiinflammatory drug (NSAID) (flunixin meglumine 1.1 to 2.2 mg/kg intravenously [IV]).[14] The decision to use perioperative and postoperative antimicrobial agents should be based on the conditions under which the surgery is performed and the diagnosis made at surgery.

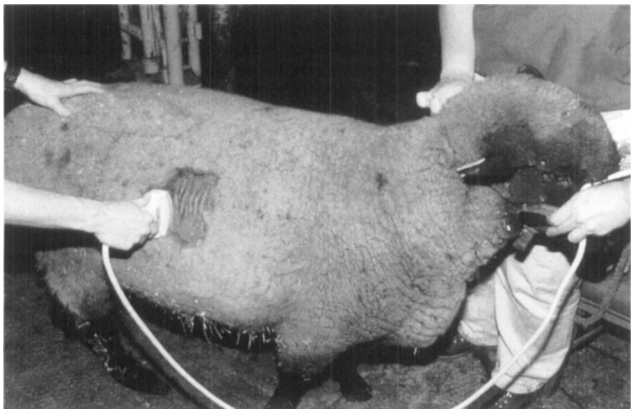

Figure 4-5 Demonstration of the site of liver ultrasonography. In sheep the area should be clipped, but in goats alcohol can be applied to the overlying hair and skin. If the area is clipped, the clinician should apply a bland coupling material (e.g., methyl cellulose, vegetable oil) between the skin and the transducer.

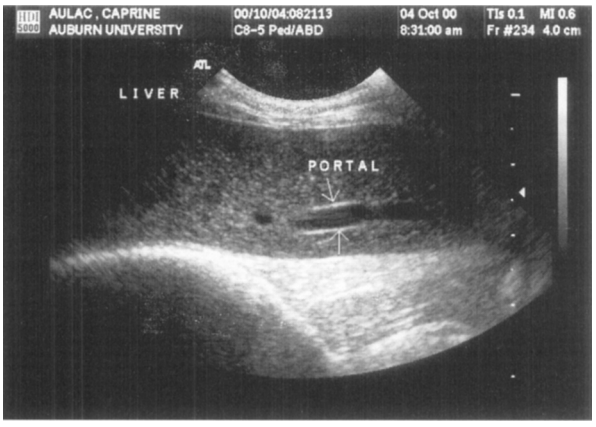

Figure 4-6 Normal ultrasonography of the liver in a goat. Note the degree of contrast. Liver abscesses, fibrosis, and fatty deposition can all be visualized.

Antimicrobial agents are not necessary for exploratory surgery performed aseptically, in a hospital setting, and without complications. However, they are indicated in field conditions, if infection is already present, and if the intestinal tract is opened. A combination of ceftiofur (1.1 to 2.2 mg/kg IV twice a day [BID]) and procaine penicillin G (22,000 IU/kg intramuscularly [IM] BID) can be administered until culture results indicate an absence of microbes.

Liver Biopsy

Liver biopsy in sheep and goats is performed using the same technique and instruments as in cattle.[12] However, sedation and ultrasound guidance are recommended.[22] The biopsy can be performed in the ninth to tenth intercostal space slightly above an imaginary line from the tuber coxae to the point of the elbow (Figure 4-7). The site should be surgically prepared, and a local anesthetic (2% lidocaine hydrochloride) infused subcutaneously. A small scalpel blade is used to make a stab incision through the skin. A 14-gauge, 11.5-cm liver biopsy instrument is inserted through the incision and the intercostal muscles and into the liver. The biopsy instrument should be directed toward the opposite elbow in most cases, but the use of real-time ultrasonography can help determine the direction and depth needed (2 to 4 cm). The clinician should avoid the vessels along the caudal border of the ribs. On reaching the liver, the clinician will note a slight increase in resistance. Samples can be submitted for culture (in a sterile plastic or glass vial or tube), histopathology (in formalin at a 10:1 ratio of formalin to tissue); and/or mineral analysis (in a plastic tube). When performing a liver biopsy for mineral analysis, the clinician should rinse the biopsy site with distilled and deionized water after sterile preparation to minimize sample contamination. Samples for mineral analysis should not be placed in formalin. The skin incision can be sutured, stapled, or, if it is small enough, left alone to heal by second intention. The clinician or keeper should apply fly repellent to the area. The animal should have a history of *Clostridium* prophylaxis; if it does not, it should be vaccinated during or before the biopsy.

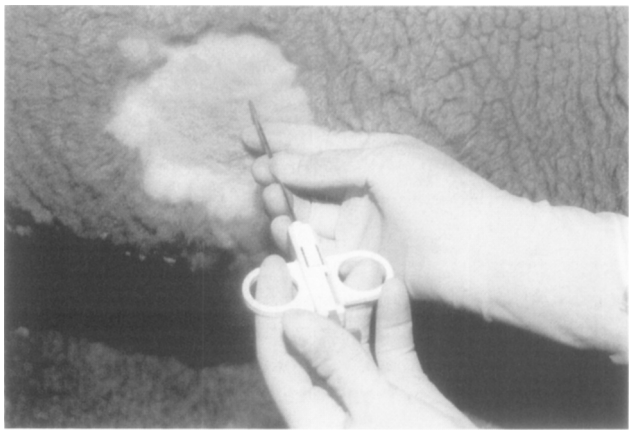

Figure 4-7 Liver biopsy. After the skin is clipped, anesthetized, and aseptically prepared, the surgeon makes a stab incision in the skin and introduces a 14-gauge biopsy needle.

References

1. *U.S. sheep health and management practices,* Fort Collins, CO, 1996, National Animal Health Monitoring System.
2. Pugh DG, Hilton CD, Mobini SM: Control programs for gastrointestinal nematodes in sheep and goats, *Comp Cont Ed Pract Vet* 20:5112, 1998.
3. Roussel AJ, Whitney MS, Cole DJ: Interpreting a bovine serum chemistry profile: Part 1, *Vet Med* 92(6):553, 1997.
4. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
5. Howard JL, Smith RA: *Current veterinary therapy 4: food animal practice,* Philadelphia, 1999, WB Saunders.
6. Howard JL: *Current veterinary therapy 3: food animal practice,* Philadelphia, 1993, WB Saunders.
7. Keneko JJ: *Clinical biochemistry of domestic animals,* San Diego, CA, 1989, Academic Press.
8. Navarre CB et al: Analysis of gastric first compartment fluid collected via percutaneous centesis from healthy llamas, *J Am Vet Med Assoc* 214(6):812, 1999.
9. Nordlund KV, Garrett EF: Rumenocentesis: a technique for collecting rumen fluid for diagnosis of subacute rumen acidosis in dairy herds, *Bovine Pract* 28:109, 1994.
10. Keefe GP, Ogilvie TH: Comparison of oro-ruminal probe and rumenocentesis for prediction of rumen pH in dairy cattle, *Proc 30th Ann Am Assoc Bovine Pract Conv,* p 168, 1997.
11. VanMetre DC, Tyler JW, Stehman SM: Diagnosis of enteric disease in small ruminants, *Vet Clin North Am: Food Anim Pract* 16:87, 2000.
12. Smith MC: Commonly encountered diseases of goats, *Proceedings of the 1996 Symposium on the Health and Disease of Small Ruminants,* 1996, Kansas City, MO.
13. Matthews J: *Colic,* ed 2, Oxford, UK, 1999, Blackwell Science.
14. House JK et al: Ancillary tests for the assessment of the ruminant digestive system, *Vet Clin North Am: Food Anim Pract* 8(2):203, 1992.
15. Tanwar RK, Saxena AK: Radiographic detection of foreign bodies (goat), *Vet Med* 79:1195, 1984.
16. Cegarra IJ, Lewis RE: Contrast study of the gastrointestinal tract in the goat *(Capra hircus), Am J Vet Res* 38:1121, 1977.
17. Braun U, Hausammann K: Ultrasonographic examination of the liver in sheep, *Am J Vet Res* 53(2):198, 1992.
18. Gourley DD, Riese RL: Laparoscopic artificial insemination in sheep, *Vet Clin North Am: Food Anim Pract* 6:615, 1990.
19. Seeger KH, Klatt PR: Laparoscopy in the sheep and goat. In Harrison RM, Wildt DE, editors: *Animal laparoscopy,* Baltimore, 1990, Williams & Wilkins.
20. Anderson DE, Gaughan EM, St-Jean G: Normal laparoscopic anatomy of the bovine abdomen, *Am J Vet Res* 54:1170, 1993.
21. Hooper RN: Abdominal surgery in small ruminants, *Proceedings of the 1998 Symposium on the Health and Disease of Small Ruminants,* 1998, Las Vegas, NV.
22. Pearson EG: Diseases of the hepatobiliary system. In Smith BP, editor: *Large animal internal medicine,* St Louis, 1996, Mosby.

DISEASES OF THE FORESTOMACHS

Bloat

Bloat is less common in small ruminants than in cattle, with goats having the condition less commonly than sheep. *Bloat* is the accumulation of either free gas or froth in the rumen, which causes rumen distention. The causes of bloat can be divided into three categories[1,2]:

1. Frothy bloat—caused by diets that promote the formation of stable froth
2. Free gas bloat—caused by diets that promote excessive free gas production
3. Free gas bloat—caused by failure to eructate

Pathogenesis. Frothy bloat is usually associated with the ingestion of legume forages or hay (particularly alfalfa) and with grazing on lush cereal grain pastures, but it also may occur with high-grain diets.[3] In the case of frothy bloat from a finely ground diet (usually corn), mucoprotein released from rumen protozoa stabilizes the foam at a low pH. In legume-associated frothy bloat, plant chloroplasts released into the rumen trap gas bubbles. Regardless of the form of frothy bloat, the small bubbles fill much of the rumen, preventing clearance of the rumen's cardia and resulting in a cessation of eructation. Free gas bloat also occurs with grain diets, especially if the animals are not adapted to the diet. Failure to eructate has a variety of causes. Physical obstructions of the esophagus such as choke or swollen mediastinal lymph nodes can cause free gas bloat. Any disease of the rumen wall can interfere with rumen contractions and eructation. Hypocalcemia, endotoxemia, pain, peritonitis, and some pharmaceutical agents (especially xylazine) can all interfere with rumen function and eructation.[1,2,4,5]

Clinical signs. Clinical signs of frothy bloat and free gas bloat from either food intake or physical obstruction of the esophagus are usually more severe and immediately life-threatening than bloat seen from rumen wall diseases and systemic influences. Abdominal enlargement occurs, particularly in the dorsal left paralumbar fossa. This may be subtle in sheep or Angora goats with full fleece. Signs of colic and anxiety are common. The rumen may be either hypomotile or hypermotile. Respiratory distress is evident, with some animals breathing through their mouths; death can ensue if the bloat is not treated.[3]

Diagnosis and treatment. This condition is a medical emergency, and therefore diagnosis and treatment should occur almost simultaneously. If the animal is not in immediate danger of dying, an orogastric tube can be passed. Most cases of free gas bloat are relieved with passage of the tube. A clinician should then take a thorough history and perform a complete physical examination to find the cause of the bloat. If the bloat is not relieved with an orogastric tube, the tube should be removed and examined for evidence of froth. Frothy bloat can be treated with poloxalene (44 mg/kg) or dioctyl sodium sulfosuccinate (DSS) (28 cc [1 oz]) delivered by orogastric tube. The froth encountered in frothy bloat caused by the ingestion of finely ground grain has a pH of less than 5.5. If frothy bloat occurs while animals are being fed concentrates, mineral oil (100 ml) may work better. Peanut oil (20 to 50 mg/kg), vegetable oil (100 to 200 ml), and hand soap (10 ml) also have been recommended in emergency situations.[3] If the animal is in severe respiratory distress, the clinician should insert a trocar or large needle into the rumen at the paralumbar fossa. If gas does not escape, or froth is seen coming out of the trocar, an emergency rumenotomy should be performed (see the Rumenotomy section of this chapter).[3] If several cases of bloat are encountered in a group of pastured animals, the entire group should be removed from the pasture and reintroduced slowly after gradual acclimation. If only one or two cases of bloat are encountered, the healthy animals can remain on the offending pasture, but grazing should be limited to ensure gradual acclimation.

Prevention. Prevention of frothy bloat involves limiting access to offending pastures or feedstuffs; providing supplemental feed and providing poloxalene in mineral supplements; and adding ionophores to the ration or supplement. When grazing or consuming legumes as "greenchop," animals should be introduced to the feed or pasture slowly, preferably over 2 to 3 weeks. Animals should be closely monitored after a frost and during the rapid growth phase of plants because legumes, particularly alfalfa, may be more likely to cause bloat at this time. Certain varieties of legumes that are designed for intensive grazing systems (e.g., Alfagraze) should be planted and managed in a manner that decreases the incidence of bloat (limited or creep grazing). Feeding dry, stemmy hay for 1 to 2 hours before allowing access to the legume pasture also may help minimize bloat. Grass-legume pastures in which legumes are limited to less than 50% of the forage are safer but can still pose a problem for animals that are selective grazers. Grazing legumes with high leaf tannin concentrations (e.g., arrowleaf clover, kudzu) is usually safer because tannins help break down rumen foam. The inclusion of poloxalene (10 to 20 mg/kg daily) in the feed or mineral supplement is useful in preventing frothy bloat. If poloxalene supplements are used, keepers should feed them for 1 to 2 weeks before moving animals onto a problem pasture.

Free gas bloat from concentrate feeds can be controlled by slow introduction to these feeds to allow for rumen

adaptation and by the inclusion of ionophores in the diet.[1] Monensin (15 mg/head/day in ewes, 1 mg/kg/day in goats) and lasalocid (0.5 to 1 mg/kg/day in sheep and goats) both decrease the formation of free ruminal gas. By enhancing propionic acid formation, these drugs not only reduce the amount of methane produced in the rumen, they also improve the efficiency of nutrient assimilation from feedstuffs.

Bloat in lambs and kids can have the same causes as in adults but also can be caused by improper milk feeding. Overfeeding, feeding of large infrequent meals, and feeding spoiled or cold milk have all been associated with bloat in lambs and kids.[6] Rapid overdistention of the abomasum and improper chemical or physical composition of milk replacers inhibit rumen motility, leading to bloat. Even though the feeding of cold milk has been associated with bloat, the practice can be used effectively in orphan feeding programs. Lambs and kids tend to limit their intake of cold milk after they have become accustomed to cold milk in a free-choice feeding system. Milk is usually placed in the rumen when animals are tube-fed; this may result in milk spoilage.[1,6]

Simple Indigestion

Simple indigestion is a mild form of upset of the reticulorumen caused by a change in feeding routine. It can be caused by an alteration in the type of feed or in the amount of feed offered. The most common causes of simple indigestion are the addition of grain to the diet, an increase in the amount of grain fed, and an increase in the energy density of the diet. Examples of such dietary changes are replacing oats with corn or changing from whole to ground corn. If the changes are drastic, rumen acidosis can occur (see the following section). Other common causes are changes in hay or pasture, consumption of moldy hay, and ingestion of weeds and toxic plants after overgrazing or droughts. Clinical signs include mild anorexia that lasts for 1 to 2 days. Mild diarrhea and bloat also may occur. Rumen fluid pH can be unchanged, increased, or decreased depending on the inciting cause. Most animals improve with no treatment.[1]

Rumen Acidosis

Pathogenesis. Rumen acidosis is caused by the rapid rumen fermentation of highly digestible carbohydrates that are ingested in excessive amounts. Although corn is commonly implicated, other cereal grains (oats, wheat, barley) may be involved, particularly if they are finely ground. The smaller the particle size, the more quickly rumen bacteria are able to ferment the carbohydrates contained in the feed. The common name of this condition is "grain overload," but breads, candy, apples and other fruits, beets, and potatoes also can cause this condition. Rumen acidosis usually occurs in animals that have been fed predominantly forage-based rations and are suddenly given access to large amounts of highly fermentable concentrates or concentrated forms of energy. It also can occur in animals that have been receiving concentrates previously, if the amount is suddenly and drastically increased; if access is denied for a time, then suddenly returned (e.g., during weather changes and alterations in water availability); or if ration mixing errors occur (e.g., leaving out monensin and rumen buffers)

As highly digestible carbohydrates are fermented, rumen pH drops. *Lactobacillus* species, which are lactic acid producers, proliferate in the acidic rumen environment and further lower rumen pH. As the rumen pH drops, rumen protozoa and many of the lactate users begin to die. Lactic acid production causes the osmotic pressure in the rumen to increase. Fluid is drawn from the systemic circulation into the rumen, resulting in dehydration and possibly hypovolemic shock. Lactate concentrations increase in the blood and may cause systemic lactic acidosis. The lactic acid in the rumen also is toxic to the rumen epithelium. Damage to the epithelium can result in leakage of bacteria and toxins into the portal and systemic circulation. Chronic sequelae to rumen acidosis include fungal rumenitis and occasionally liver abscesses.[1,7] Liver abscesses are less commonly encountered in sheep and goats than in cattle. Laminitis also can occur, but may be more of a problem in sheep than in goats.[8] The severity of the disease depends on the composition of the feed, particle size, amount of feed consumed, and the period of adaptation to the diet.

Clinical signs. Clinical signs vary with the amount and type of feed ingested and the time since ingestion. Signs first appear 12 to 36 hours after ingestion of the offending feed; they vary from anorexia, depression, and weakness to a down animal suffering from severe circulatory shock. Dehydration is usually severe and evidence of toxemia is present (e.g., injected mucous membranes, increased scleral injection). Colic, bilateral ventral abdominal distention, rumen stasis, and a "splashy" feel to the rumen also may be present. Diarrhea can develop, adding to dehydration.[1,8,9] The diarrhea can range from a pastelike feces to very watery droppings with foam and occasionally pieces of grain easily recognized. Dehydration, lactic acidosis, and toxemia result in neurologic signs, including ataxia, head pressing, opisthotonos, and seizures. The body temperature is initially elevated but may drop as the condition worsens or the animal becomes toxic. Some animals develop polioencephalomalacia and appear blind.

Diagnosis. The rumen fluid pH may fall below 5.5. The fluid itself is milky gray and particles of the inciting

feed may be noticed. Protozoa are usually reduced in number or absent, and large gram-positive rods (*Lactobacillus* species) may be seen on Gram's stain.[9] Clinicopathology is consistent with dehydration (increased PCV and total protein, prerenal azotemia) and metabolic acidosis.[9] Liver enzymes (gamma-glutamyl transpeptidase [GGT], aspartate aminotransferase [AST], lactate dehydrogenase [LDH]) may be elevated on serum biochemical analysis.[1,10] The leukogram can vary from normal to a degenerative left shift, depending on the severity of the case. Urinalysis reveals an increased specific gravity.

Treatment. Treatment is aimed at correcting cardiovascular shock, dehydration, acidosis, and toxemia and removing or neutralizing the offending feedstuffs. IV fluids containing 5% sodium bicarbonate should be administered.[1,11] Oral fluids are contraindicated because they cannot be absorbed and may increase the rumen distention and discomfort of the animal. NSAIDs are indicated for toxemia (flunixin meglumine, 1.1 to 2.2 mg/kg IV).[1,11] Oral administration of magnesium hydroxide and magnesium oxide (1 g/kg) may neutralize the acidic pH and is sufficient in mild cases. However, if much of the feed is still in the rumen, these two alkalinizing agents will only work temporarily. Oral antibiotics have been recommended to kill rumen microflora and stop fermentation. However, the authors of this chapter feel they are contraindicated because the gram-negative anaerobes that need to flourish to reestablish normal rumen microflora are susceptible to most antimicrobials effective against *Lactobacillus* species. Removing the substrate for the *Lactobacillus* species is more effective. Because orogastric tubes with large enough bores to reflux feedstuffs are too large for sheep and goats, rumenotomy is indicated in severe cases to remove the feed (see the section on Rumenotomy in this chapter). After the rumen pH is corrected, transfaunation of the rumen microflora with about 1 qt of rumen fluid from a small ruminant is beneficial (Box 4-2). Thiamine supplementation (vitamin B_1, 5 mg/lb subcutaneously [SC] three times a day [TID] to four times a day [QID]) is indicated until rumen function returns.[11] In certain instances, calcium may be indicated and can be included in the IV fluids (calcium gluconate). The clinician should avoid mixing calcium salts and sodium bicarbonate. Bacterial leakage into the rumen wall, liver, and systemic circulation makes antimicrobial therapy necessary. The systemic antimicrobial agent of choice is penicillin (procaine penicillin G, 22,000 IU/kg IM BID) because anaerobes are the most likely offending organisms. If treated aggressively, the prognosis for immediate survival is good. Feed (grass hay only) and water should be limited until rumen contractions return to prevent overdistention of the rumen. The chronic sequelae discussed previously influence long-term survival.

Prevention. Prevention involves introducing concentrate feeds slowly to allow rumen microflora adaptation. Dietary change from a lower to a higher fermentable energy concentration should occur slowly and preferably over a 2- to 3-week period. In the case of animals being fed high-grain rations (e.g., club lambs, feedlot lambs, dairy goats), buffering agents can be added to the diet. Rumen buffers may improve milk production, increase feed intake, and increase rate of gain. The crude fiber content should comprise a minimum of 20% of the diet's total digestible nutrients (TDN). For example, the TDN is 75%, the minimum acceptable crude fiber is 15%. Crude fiber levels lower than this can be fed for short periods if the rumen is properly adapted, but problems may nevertheless occur. Sodium bicarbonate is probably the most commonly used buffer; it can be offered free choice or included in the diet as 1% of dry matter intake. Calcium carbonate or limestone (which both have low

BOX 4-2

Collection, Handling, and Storage of Rumen Fluid for Transfaunation

Collection	Collection is easiest from the rumen of a fistulated adult cow. If a fistulated cow is unavailable, fluid can be collected through a weighted orogastric tube. Alternatively, fluid can be collected from any normal ruminant at slaughter.
Handling	Rumen contents collected from a fistulated cow or at slaughter can be strained through gauze or cheesecloth to separate the fluid from the fibrous contents. Fluid collected through a weighted tube should be ready for storage.
Storage	Rumen fluid should ideally be administered immediately. However, it can be stored for 24 to 48 hours. The surface of the fluid should be covered with a layer of mineral oil to maintain an anaerobic environment and stored at refrigerator temperature. CAUTION: Do not store rumen fluid in a closed container because it may explode.

rumen solubility) and magnesium oxide (which has poor palatability) also can be included in the feed. Magnesium oxide should be limited to 0.5% to 0.8% of the dry matter intake.

Reticulitis/Rumenitis/Parakeratosis

Pathogenesis. Reticulitis and rumenitis can result from chemical or mechanical damage to the mucosal lining of the reticulorumen. The most common cause of chemical damage is rumen acidosis. However, ingestion of caustic toxins also can damage the mucosa. Mechanical damage can occur from ingested foreign bodies or the formation of rumen bezoars. In cattle, some viruses such as the ones that cause bovine virus diarrhea and infectious bovine rhinotracheitis can infect the rumen wall. Similar viruses have yet to be identified in sheep and goats. After the mucosa has been damaged, secondary infection by bacteria or fungi can occur.[12] Previous treatment with oral antibiotics may predispose to fungal infections of the rumen wall, especially if the mucosa is already damaged. Actinobacillosis, actinomycosis, and tuberculosis rarely affect the rumen wall. Tumors of the rumen wall also have been reported.[1,13] Not all of these causes of reticulitis and rumenitis have been reported in sheep and goats, but all are potential problems.

Clinical signs. The clinical signs of these diseases are vague. Anorexia and forestomach hypomotility may be the only clinical signs.

Diagnosis. Confirming a diagnosis also may prove difficult. Samples of rumen fluid may only show changes associated with anorexia (alkaline pH, decreased numbers and motility of protozoa, prolonged MBR time; see Table 4-1 for normal values). Occasionally fungal organisms may be seen on Diff Quik stained slides of rumen fluid. In these cases a diagnosis of fungal rumenitis should be made. An exploratory laparotomy and rumenotomy may be required to diagnose foreign bodies or masses. Rumen parakeratosis is characterized by dark, thickened, and clumped rumen papillae. It is seen mainly in feedlot lambs that consume finely ground or pelleted rations.[14] The parakeratotic rumen papillae are fragile and predisposed to damage, which can increase the chances of rumenitis.[1]

Treatment and prevention. Treatment depends on the inciting cause. Dietary changes should be made to decrease energy density and increase fiber intake. Mild rumenitis may improve with time and supportive care (transfaunation, fluid support, high-quality feed). Fungal rumenitis can be treated with thiabendazole (25 mg/kg orally).[15] Severe changes may lead to scarring and permanent impairment of rumen function.

DISEASES OF THE RETICULORUMEN

Traumatic Reticuloperitonitis

Traumatic reticuloperitonitis is not as common in small ruminants as in cattle, but it has been reported. Goats are affected more commonly than sheep. This is probably because of the dietary habits of small ruminants; they tend to be selective grazers and do not "vacuum" the ground as cattle do. Offending foreign bodies that cause traumatic reticuloperitonitis include pieces of wire and needles.[16,17] The clinical signs are identical to those in cattle and may include anorexia, depression, colic, signs of heart failure, and evidence of draining tracts from the chest cavity. Treatment is usually difficult.

Rumen Impaction

Rumen impaction can occur after dehydration, blockage of the omasal orifice by a foreign body, sand ingestion, or consumption of diets high in fiber and low in digestibility.[18] Clinical signs are nonspecific, but the firm rumen can usually be palpated in the left flank. The feces may be scant and dry. Oral fluids containing magnesium sulfate (60 g) may loosen impactions, but a rumenotomy is required in severe cases.[18]

Rumenotomy

To reduce rumen fill, sheep or goats should ideally have feed withheld for 24 hours before rumenotomy. However, this is usually impossible because in most cases rumenotomy is an emergency procedure. The perioperative administration of antimicrobial agents is essential because even with meticulous technique some contamination of the incision site and possibly the peritoneal cavity is inevitable. Because the rumen microflora is predominantly composed of anaerobic bacteria, penicillin (22,000 IU/kg) is the antimicrobial agent of choice and should be administered 2 to 4 hours before surgery. If the rumenotomy is being performed in an emergency situation, penicillin salts (potassium or sodium) that can be given IV provide therapeutic concentrations more rapidly than procaine penicillin. NSAIDs (flunixin meglumine, 1.1 to 2.2 mg/kg IV) also are recommended before surgery. If necessary, treatment of cardiovascular shock and dehydration with IV fluids also should begin before surgery and continue until the animal is rehydrated and in stable condition (see Appendix II).

General anesthesia is recommended, but heavy sedation and local anesthetic infiltration of the incision site can be efficiently used (see Chapter 16). The clinician should clip and surgically prepare a square area from 5 cm in front of the last rib to the tuber coxae, and from the

dorsal midline to the lower abdomen, encompassing the entire left paralumbar fossa.

The surgeon makes a skin incision approximately 5 cm longer than the width of the hand 5 cm caudal and parallel to the last rib. The incision is continued through the muscle layers into the abdomen. Because the abdominal wall is relatively thin, the surgeon should take care not to enter the rumen or bowel. The surgeon grasps the rumen wall and pulls it through the incision; suturing it to the skin with a simple continuous circular pattern around the entire incision. This forms a seal that minimizes rumen content contamination of the deep layers of the incision and peritoneal cavity. The rumen wall is then incised inside the circle of sutures. The incision in the rumen wall should be large enough for the surgeon to put his or her hand inside the rumen without traumatizing the rumen wall.

After the rumen has been explored and emptied and the primary reason for doing the procedure has been completed, the surgeon closes the rumen wall in a continuous inverting pattern (Cushing, Lembert, or Guard's rumen stitch) with absorbable suture (0 catgut). The area should be rinsed with copious amounts of sterile isotonic fluids, and a new set of sterile instruments, sterile gloves, and surgical attire should be used for the remainder of the surgery. The surgeon then removes the suture securing the rumen to the skin and rinses the area again before performing routine closure of the abdominal muscles and subcutaneous layers with absorbable suture (0 catgut) in simple continuous patterns, taking care to close dead space between layers. The skin is closed with a continuous pattern (Ford interlocking) using a nonabsorbable suture material.

The sheep or goat should be observed closely by the clinician for signs of complications, including peritonitis, incisional dehiscence, incisional hematoma, abscess, and hernia formation. Penicillin therapy (procaine penicillin G, 22,000 IU/kg BID) should continue for at least 5 days. The skin sutures can be removed 10 to 14 days after surgery.

References

1. Garry FB: Indigestion in ruminants. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
2. Guard C: Bloat or ruminal tympany. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis 1996, Mosby.
3. Matthews J: *Diseases of the goat,* ed 2, Oxford, UK, 1999, Blackwell Science.
4. Brikas P, Tsiamitas C, Wyburn RS: On the effect of xylazine on forestomach motility in sheep, *J Vet Med* 33:174, 1986.
5. van Miert ASJPAM, van Duin CTM, Anika SM: Anorexia during febrile conditions in dwarf goats: the effect of diazepam, flurbiprofen and naloxone, *Vet Quart* 8:266, 1986.
6. Chennells D: Bloat in kids, *Goat Vet Soc J* 2:16, 1981.
7. Nour MSM, Abusamra MT, Hago BED: Experimentally induced lactic acidosis in Nubian goats: clinical, biochemical, and pathological investigations, *Sm Rumin Res* 31(1):7, 1999.
8. VanMetre DC, Tyler JW, Stehman SM: Diagnosis of enteric disease in small ruminants, *Vet Clin North Am: Food Anim Pract* 16:87, 2000.
9. Braun U, Rihs T, Schefer U: Ruminal lactic acidosis in sheep and goats, *Vet Rec* 130(16):343, 1992.
10. Lal SB et al: Biochemical alterations in serum and cerebrospinal fluid in experimental acidosis in goats, *Res Vet Sci* 50(2):208, 1991.
11. Smith MC: Commonly encountered diseases of goats, *Proceedings of the 1996 Symposium on the Health and Disease of Small Ruminants,* 1996, Kansas City, MO.
12. Perez V et al: Generalized aspergillosis in dairy sheep, *J Vet Med* 46(9):613, 1999.
13. Norval M et al: Rumen papillomas in sheep, *Vet Microbiol* 10(3):219, 1985.
14. Kutas F, Galfi P, Neogrady S: Effect of monensin on development of ruminal parakeratosis in fattening lambs, *Zentralblatt fur Veterinarmedizin* 30(7):506, 1983.
15. Kersting KW, Thompson JR: Lactic acidosis. In Howard JL, Smith RA, editors: *Current veterinary therapy 4: food animal practice,* Philadelphia, 1999, WB Saunders.
16. Sharma KB, Ranka AK: Foreign body syndrome in goats—a report of five cases, *Indian Vet J* 55(5):413, 1978.
17. Maddy KT: Traumatic gastritis in sheep and goats, *J Am Vet Med Assoc,* p 124, Feb 1954.
18. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.

DISEASES OF THE ABOMASUM

Abomasitis

Abomasitis and abomasal ulcers in adult sheep and goats are associated with rumen acidosis or chronic rumenitis but also can be caused by infections.[1-4] Finely ground feeds, pelleted rations, systemic stress, and feeding lush forages have all been implicated. Anecdotal associations with mineral deficiency (copper) have gone unproved.

Clinical signs and diagnosis. This disease often goes unnoticed in mild cases, and the most common signs are anorexia and colic. No definitive antemortem diagnostic tests are available. Fecal occult blood is often absent. Occasionally dark stool, altered appetite (wood chewing), and bruxism are seen. Therefore other causes of colic should be eliminated. Diagnosis is based on clinical signs.

Treatment. Effective therapy can be difficult. Oral medications such as coating agents must first pass through the rumen, and therefore arrive at the abomasum diluted. IV (not oral) ranitidine (15 mg/kg once a day [SID]) may be beneficial.[5] Herd problems of rumen acidosis may be addressed with buffers in the feed.

Abomasal Hemorrhage

A syndrome of abomasal hemorrhage, bloat, and ulceration is seen in lambs and kids 2 to 10 weeks of age. *Sarcina*-like bacteria, *Clostridium falax, Clostridium sordelli,* and *Clostridium septicum* have been isolated from

many of these cases.[6-9] *C. septicum* infections of the abomasum are commonly called *braxy.*[1] The feeding of milk replacer free choice, iron deficiency, and bezoars have been implicated as predisposing factors.[10,11]

Clinical signs. The signs of this syndrome are severe, acute abdominal distention; colic; and death.[6-9]

Diagnosis and treatment. The diagnosis of this condition is by postmortem examination. Treatment in suspected antemortem cases is unsuccessful.

Prevention. Adding formalin to milk replacers and vaccinating for clostridial diseases may decrease the occurrence of this disease.[10,12] Lambs or kids on problem farms can be vaccinated for *Clostridium* species during the first week of life with multivalent bacterins.

Abomasal Impaction

Similar to rumen impaction, abomasal impaction usually occurs when poor-quality roughage is fed, but it also can be seen with foreign body obstruction of the pylorus.[4,13,14] Goats appear to be more commonly affected than sheep, and Boer goats are more commonly affected than Angora goats.[15] Pregnant animals may be more prone to this condition.

Clinical signs and diagnosis. Affected animals are usually anorexic. They have mild distention of the ventral abdomen, and in some cases the firm abomasum can be palpated through the abdominal wall on the right side.[16] Weight loss may be apparent. Clinicopathologic evaluation may be normal, or mild hypochloremic metabolic alkalosis may be present, with elevated rumen chloride concentrations (more than 50 mEq/L).[16]

Treatment. Diet changes and mineral oil by mouth (PO) are the most commonly employed treatments. Abomasotomy can be attempted, but it has rarely been reported in small ruminants and does not usually improve the animals' long-term prognosis. When attempting abomasotomy, the clinician should perform the procedure with the animal in dorsal recumbency and under general anesthesia. The abomasum can best be visualized through an incision parallel and to the right of midline, caudal to the xyphoid process. The prognosis is poor.[13]

Prevention. Dietary manipulation to improve feed or forage quality is the best mode of prevention.

Abomasal Emptying Defect

Abomasal emptying defect is a disease that presents similarly to abomasal impaction but is recognized only in Suffolk sheep. The underlying cause is unknown. Unlike abomasal impaction, this disease is associated with concentrate feeding and often occurs around lambing time. The clinical signs are chronic weight loss, abdominal distention, and anorexia. Clinical pathology and rumen chloride levels are the same as described for abomasal impaction. On necropsy the abomasum is greatly distended, and the contents may be liquid or dry. Treatment with laxatives, cathartics, motility modifiers, and abomasotomy has been mostly unsuccessful.[17-19]

Azalea, Laurel, and Rhododendron Toxicity

Members of the azalea, laurel, and rhododendron plant group produce andromedotoxins that alter sodium metabolism, resulting in prolonged nerve depolarization. These plants are cardiotoxic, but affected animals generally exhibit acute gastrointestinal upset. These evergreen shrubs produce thick, dark green leaves. They also have five-lobed, white to pink, saucer-shaped flowers that bloom around July. Some of these plants are grown as ornamental shrubbery around homes, whereas others grow wild along streams, cliffs, and rocky slopes. They can be short or tall (as large as 10 m) and can form thickets. All parts of these plants are toxic.

Clinical signs. Animals browsing a new area, those fed clippings from trimmed azalea hedges, and underfed, hungry animals given access to these plants are likely candidates for intoxication. Animals that ingest as few as two or three leaves may show signs of salivation, grinding teeth, nasal discharge, colic, epiphora, and acute digestive upset within 6 hours of ingestion. As the intoxication progresses, animals become depressed and exhibit projectile vomiting, frequent defecation, and a slowed pulse. Terminally intoxicated animals become paralyzed and comatose. Some sheep and goats develop aspiration pneumonia secondary to intoxication.

Diagnosis. The diagnosis of this condition is usually based on clinical signs coupled with a history of ingestion of one of these plants and/or the discovery of these plants in the gastrointestinal tract.

Treatment. Intoxicated animals may recover in 1 to 2 days without any therapy if the offending plants are removed from the diet. However, the administration of charcoal (2 to 9 g/kg PO), atropine (0.06 to 0.1 mg/kg IV), other antiarrhythmic drugs, and IV fluids all may be indicated. To manage the aspiration pneumonia, the administration of antibiotics (penicillin 22,000 units/kg BID IM) and oral magnesium hydroxide also may be beneficial. Obviously, any existing dehydration should be corrected (see Appendix II).

Prevention. Mountainous or hilly areas should be fenced. Feeding shrubbery clippings is discouraged.

References

1. Kimberling CV: *Jensen and Swift's diseases of sheep,* ed 3, Philadelphia, 1988, Lea & Febiger.
2. Matthews J: *Diseases of the goat,* ed 2, Oxford, UK, 1999, Blackwell Science.
3. Gundula A, Shirley H: Two cases of phycomycotic ulceration in sheep, *Vet Rec* 77:675, 1965.
4. Linklater KA, Smith MC: *Color atlas of diseases and disorders of the sheep and goat,* London, 1993, Wolfe Publishing.
5. Duran SH et al: pH changes in abomasal fluid of sheep treated with intravenous and oral ranitidine, *Proceedings of the Eleventh ACVIM Forum,* Washington, DC, 1993, American College of Veterinary Internal Medicine.
6. DeBey BM, Blanchard PC, Durfee PT: Abomasal bloat associated with *Sarcina*-like bacteria in goat kids, *J Am Vet Med Assoc* 209(8):1468, 1996.
7. Vatn S, Tranulis MA, Hofshagen M: *Sarcina*-like bacteria, *Clostridium falax* and *Clostridium sordelli* in lambs with abomasal bloat, haemorrhage and ulcers, *J Comp Path* 122(2/3):193, 2000.
8. Ellis TM, Rowe JB, Lloyd JM: Acute abomasitis due to *Clostridium septicum* infection in experimental sheep, *Aust Vet J* 60(10):308, 1983.
9. Eustis SL, Bergeland ME: Suppurative abomasitis associated with *Clostridium septicum* infection, *J Am Vet Med Assoc* 178(7):732, 1981.
10. Vatn S, Ulvund MJ: Abomasal bloat, haemorrhage and ulcers in young Norwegian lambs, *Vet Rec* 146(2):35, 2000.
11. Vatn S, Torsteinbo WO: Effects of iron dextran injections on the incidence of abomasal bloat, clinical pathology and growth rates in lambs, *Vet Rec* 146:462, 2000.
12. Gorrill AO, Nicholson JWG, MacIntyre TM: Effects of formalin added to milk replacers on growth, feed intake, digestion and incidence of abomasal bloat in lambs, *Can J Anim Sci* 55(4):557, 1975.
13. Bath GF, Bergh T: A specific form of abomasal phytobezoar in goats and sheep, *J South African Vet Med Assoc* 50(2):69, 1979.
14. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
15. Bath GF: Abomasal phytobezoariasis of goats and sheep, *J South African Vet Med Assoc* 49:133, 1979.
16. Kline EE et al: Abomasal impaction in sheep, *Vet Rec* 113(8):177, 1983.
17. Guard C: Abomasal dilation and emptying defect of Suffolk sheep. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
18. Rings DM et al: Abomasal emptying defect in Suffolk sheep, *J Am Vet Med Assoc* 185(12):1520, 1984.
19. Ruegg PL, George LW, East NE: Abomasal dilation and emptying defect in a flock of Suffolk ewes, *J Am Vet Med Assoc* 193(12):1534, 1988.

DISEASES OF THE INTESTINES

Diarrhea in Lambs and Kids

Diarrhea in lambs and kids is a complex, multifactorial disease involving the animal, the environment, nutrition, and infectious agents. Decades of research have been devoted to the study of the pathophysiology of infectious diarrhea of calves; the pathology in lambs and kids is quite similar. Despite improvements in management practices and prevention and treatment strategies, diarrhea is still the most common and costly disease affecting neonatal ruminants.[1-4]

Some general preventive measures (e.g., improved sanitation) decrease disease no matter the cause. However, specific control measures such as vaccination require the definition of a specific cause of diarrhea. Table 4-2 lists the agents most likely to cause diarrhea in lambs and kids, tissues or other samples required for diagnosis, and commonly employed test methods. The color and consistency of the feces and any gross lesions can appear similar no matter the cause. Therefore laboratory identification of infectious agents and tissue histopathology are key to establishing a diagnosis. Because autolysis and secondary bacterial invasion of the gut begins within minutes of death, necropsy samples taken immediately from euthanized lambs and kids yield the most reliable diagnostic material. Mixed infections with two or more pathogens are common, and pathogens that are a problem on a farm change from year to year.[3,5,6] In some cases an underlying nutritional deficiency or excess may occur concurrently with an infectious agent. Therefore the clinician should be careful to take a variety of samples to ensure that all pathogens and predisposing factors involved are recognized; continued reevaluation of the causes of diarrhea is crucial. Examination of several cases, with a focus on those in the acute phases, is important. Although examination of antemortem fecal samples can be diagnostic, laboratory testing of tissue samples may yield better results. Treatment and preventive measures specific to a particular disease are discussed with that disease in the following paragraphs. General supportive treatment and control measures are covered at the end of this section.

Causes of Diarrhea in Neonatal Lambs and Kids

Four major pathogens cause diarrhea in lambs and kids during the first month of life: enterotoxigenic *Escherichia coli* (ETEC), rotavirus, *Cryptosporidium* species, and *Salmonella* species. The relative prevalence of these infectious agents varies greatly among studies. This variance most likely results from differences in location, season, diagnostic techniques, and the occurrence of mixed infections. Other, less common causes of diarrhea in neonates are *Giardia* infections and nutritional diarrhea. Figure 4-8 shows the ages at which diarrhea is expected with certain infections.

Enterotoxigenic *Escherichia coli*

Pathogenesis. ETEC employs two virulence factors to cause disease. The first is the ability to attach and colonize the intestinal villi, which is accomplished via fimbria or pili. The most important fimbria in lambs are K99 and F41.[7,8] The fimbrial antigens can be recognized from samples sent to most diagnostic laboratories and are im-

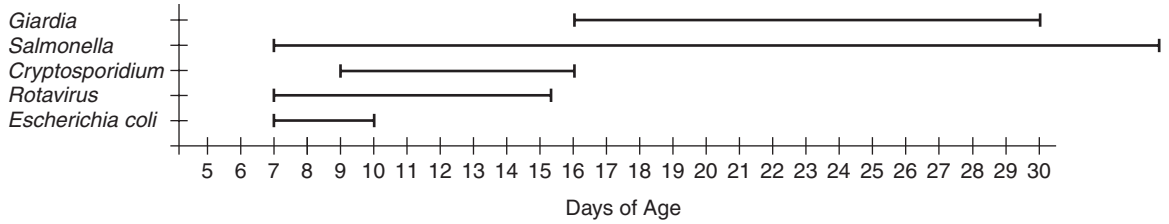

Figure 4-8 Ages at which infectious agents cause diarrhea in lambs and kids.

TABLE 4-2

DIAGNOSTIC SAMPLES AND TESTING METHODS REQUIRED FOR DIFFERENTIATION OF THE MOST COMMON CAUSES OF INFECTIOUS DIARRHEA OF LAMBS AND KIDS

CAUSATIVE AGENT	SAMPLE REQUIRED	TEST METHOD*
Escherichia coli	2 to 3 g feces	Culture and serotyping for K99 and F41
	Formalin-fixed small intestine	Histopathology
Rotavirus	2 to 3 g feces or colonic contents	EM, ELISA, VI, CF, PCR
	Formalin-fixed small and large intestine	Histopathology
	Frozen small and large intestine	VI, FA, IP
Cryptosporidia	2 to 3 g feces	FA, fecal flotation
	Air-dried fecal smear	Acid-fast stain
	Formalin-fixed small and large intestine	Histopathology
Salmonella	2 to 3 g feces	Culture, PCR
	Formalin-fixed small and large intestine	Histopathology
	Frozen small and large intestine and mesenteric lymph nodes	Culture
Giardia	Wet mount of feces	Iodine staining
	Feces	ELISA, FA
Clostridium perfringens	Frozen small intestinal contents and abomasum, small and large intestine	Culture, toxin identification
	Formalin-fixed abomasum and small and large intestine	Histopathology
Coccidia	2 to 3 g feces	Fecal flotation
	Formalin-fixed small and large intestine	Histopathology

From Rings DM, Rings MB: Managing *Cryptosporidium* and *Giardia* infections in domestic ruminants, *Vet Med* 91(12):1125, 1996; Cohen ND et al: Comparison of polymerase chain reaction and microbiological culture for detection of salmonella in equine feces and environmental samples, *Am J Vet Res* 57:780, 1996; Drolet R, Fairbrother JM, Vaillancourt D: Attaching and effacing *Escherichia coli* in a goat with diarrhea, *Can Vet J* 35(2):122, 1994.

**EM,* Electron microscopy; *ELISA,* enzyme-linked immunospecific assay; *VI,* virus isolation; *CF,* complement luxation; *PCR,* polymerase chain reaction; *FA,* fluorescent antibody; *IP,* immunoperoxidase.

portant in diagnosing this agent as a cause of diarrhea. After the organism attaches to the villi, it produces the second virulence factor, enterotoxin. Enterotoxin interferes with the normal physiology of the gut, with resultant diarrhea.[8] Calves have an age-associated resistance, most likely related to the blocking of fimbrial attachment to the gut, so ETEC occurs mainly in calves less than a week old.[9,10] The mode of infection is fecal-oral.

Clinical signs. ETEC is seen in lambs and kids less than 10 days of age but is most common at 1 to 4 days of age, so age-related resistance also may occur in these animals.[3,7] It usually presents as an outbreak in lambs and kids between 12 and 48 hours of age. Because ETEC causes a "secretory" diarrhea, bicarbonate loss in the diarrhea leads to severe acidosis, with lambs and kids quickly becoming dehydrated and recumbent. However, many infected animals die before developing diarrhea. Affected neonates are depressed, stop nursing, and may show excessive salivation. Fluid sequestration in the abomasum causes a "splashing" sound on movement. This condition results in high mortality if animals are not treated promptly.[7]

Diagnosis. Fecal culture and serotyping for the K99 and F41 fimbrial antigens are the basis for diagnosis. Because many nonpathogenic *E. coli* are normal gut inhabitants, simply culturing this organism is usually insignificant.[8] Occasionally the bacteria do not express the fimbrial antigens in culture, so ETEC cannot be ruled out if the culture is negative for K99 and F41.[11] Histologic evidence of colonization of the small intestine can support a diagnosis.

Treatment. Supportive care consisting of fluid therapy with either oral, IV, or SC administration of a polyionic solution is the mainstay of therapy. The use of oral antimicrobial agents is controversial. Although antibiotics may kill the ETEC, they also may interfere with normal gut flora. If fluid support is provided, the diarrhea usually subsides without antibiotic treatment. Still, oral neomycin (10 to 12 mg/kg BID) or trimethoprim sulfa (30 mg/kg PO) and systemic ampicillin (10 to 20 mg/kg IM BID) or amoxicillin (10 to 20 mg/kg IM TID) may be beneficial. NSAIDs are indicated to decrease inflammation of the gut and provide some analgesia. The use of flunixin meglumine (1 to 2 mg/kg IM) has been shown to decrease fecal output in ETEC infections in calves[12] and appears to be beneficial in lambs.

Prevention. It is recommended that clinicians vaccinate ewes and does with bovine ETEC vaccine before they give birth to increase passive immunity.[3,4,8] Monoclonal and polyclonal antibody products for calves may be beneficial during an outbreak if it can be given to lambs or kids within the first 12 hours of life. The use of neomycin (10 to 12 mg/kg PO BID) in lambs that appear normal may help stop the progression of an outbreak. Shearing ewes prepartum to minimize fecal ingestion by neonates and ensuring that newborns ingest adequate colostrum both help decrease the incidence of this disease. Making sure that ewes and does give birth at a 2.5 to 3.5 body condition score increases the chance of adequate colostrum manufacture by the dam.

Rotavirus

Pathogenesis. Lambs and kids are infected with group B rotaviruses, whereas most other animals and human beings are infected with group A rotaviruses.[13] Rotaviruses infect villus tip cells of the small intestine, which results in villus atrophy and malabsorptive diarrhea.[14]

Clinical signs. Rotavirus generally causes diarrhea in lambs and kids 2 to 14 days old, but older animals also can be affected. Young animals can become very depressed and dehydrated.[3,13,15,16]

Diagnosis. Detection of the organism by electron microscopy of fecal or colonic samples or by immunologic techniques on feces or tissue sections is the basis of diagnosis.[13,16] Because these organisms are sloughed with the villus tip cells they infect, and viral antigens are complexed with the lambs' and kids' antibodies, tissue samples from acutely infected animals are best.[17] Rotavirus has been detected in animals without diarrhea, so other causes of diarrhea should be investigated as well.[4,5]

Treatment and prevention. Rotavirus is treated with supportive care. Prevention by vaccination of ewes and does with bovine rotavirus vaccines before they give birth is recommended to increase passive immunity.[3,4,8]

Cryptosporidiosis

Pathogenesis. *Cryptosporidium parvum* is a protozoa that can cause a malabsorptive diarrhea similar to that seen with rotavirus infection. Unlike other protozoal agents such as the one that causes coccidiosis, cryptosporidia do not require fecal excretion for sporulation to infective stages.[18] They sporulate in the gut and about 20% become immediately infectious to other villus tip cells without ever leaving the intestines. This method of autoinfection can result in severe disease that can be sustained for long periods. Because some of the oocysts also are immediately infectious when they are shed in feces, spread of infection can occur quickly.

Clinical signs. Cryptosporidia can cause diarrhea in lambs and kids 5 to 10 days of age.[4,19,20] Affected animals are often active, alert, and nursing. The diarrhea is usually very liquid and yellow. Diarrhea can vary from mild and self-limiting to severe, especially with mixed infections.[4,5,19,21] Relapses are quite common, and this organism usually occurs as a component of mixed infections.

Diagnosis. Acid-fast staining of air-dried fecal smears is a quick and easy method of diagnosis. Examination under 40× to 100× magnification reveals round protozoa that have taken up the red color of the carbol fuchsin portions of the stain on a green background (Figure 4-9). Although they can be diagnosed by fecal flotation, their very small size (4-6 μm) makes this method difficult and subject to false negative results.[22,23] Both immunologic and polymerase chain reaction (PCR) techniques have been developed to improve detection limits.[22,24] Cryptosporidia also can be identified with histology. Cryptosporidiosis is a zoonotic disease, and people can easily become infected from handling infected animals or feces.[18]

Prevention. No consistently effective treatment for cryptosporidiosis in ruminants has been identified. Anecdotal reports suggest that decoquinate and monensin sodium may be useful in control of cryptosporosis. Decoquinate (2.5 mg/kg PO) may be very useful in prevention of cryptospirosis in goats and possibly kids. During an

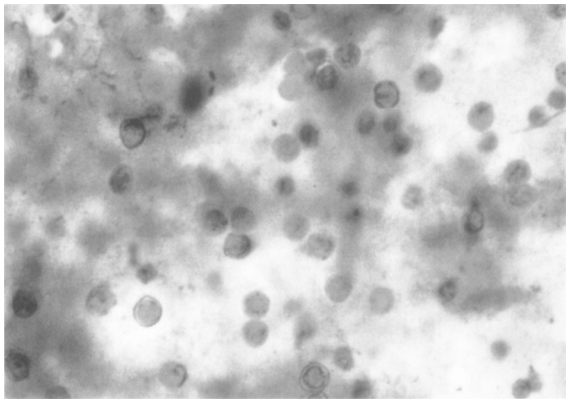

Figure 4-9 Red-staining *Cryptosporidium* on a blue-green background in a fecal smear prepared with an acid-fast stain. This protozoal parasite induces villous atrophy and decreased digestion.

outbreak affected animals should be isolated from the rest of the flock. No new animals should be added to a pen in which the disease has been diagnosed. Keepers should depopulate pens in which the disease has been diagnosed and attempt to clean the environment. Cryptosporidiosis can be particularly difficult to control because of the organisms' persistence in the environment and resistance to most chemical disinfectants. However, ammonia (5% to 10%) and formalin (10%) seem to be most effective.[19,25] Feeders should be constructed to minimize fecal contamination. Studies are currently underway to develop a vaccine for cryptosporidiosis in cattle. Early results are favorable, and this may prove the best way to control the disease in the future.[26] This is potentially a zoonotic disease, and therefore clinicians and keepers should exercise great caution when handling affected animals.

Salmonellosis

Pathogenesis. The bacterial genus *Salmonella* has thousands of serotypes, and all can potentially cause diarrhea in animals. *Salmonella* can cause diarrhea in lambs and kids of any age.[3,4] The microbes produce enterotoxins, are invasive, and cause severe inflammatory disease and necrosis of the lining of the small and large intestines.

Clinical signs. Animals less than 1 week old are more likely to die acutely without clinical signs, whereas animals older than 1 week are more likely to have diarrhea.[4,7,27] An acute onset of fever, depression, tenesmus, and shock is occasionally observed. *Salmonella*-induced diarrhea is more likely to contain blood.[4] This also is a zoonotic disease that warrants protective measures.

Diagnosis. A diagnosis of this condition is based on culture of the organism in feces or tissues and histologic examination of the small and large intestine.[28] More sensitive PCR techniques for identifying *Salmonella* species in feces are being developed.[29] The diarrhea may occasionally contain fibrin, but many animals die before this is observed. Clinicians may note leukopenia or leukocytosis in the CBC results.

Treatment. Therapy for *Salmonella*-induced diarrhea involves supportive care and possibly parenteral antimicrobial therapy. The use of antimicrobial agents is controversial and probably does not influence the gastrointestinal infection. However, because this is an invasive organism, parenteral use of antimicrobial agents may be beneficial in preventing septicemia. Antimicrobial susceptibility patterns are difficult to predict for *Salmonella* species, so antimicrobial therapy should be based on culture and sensitivity results. Ceftiofur sodium (1.1 to 2.2 mg/kg IM BID) or trimethoprim sulfadiazine (15 mg/kg SC SID) can be administered until antimicrobial sensitivity results are known.

Prevention. Latent carriers of *Salmonella* can potentially shed organisms to other animals, particularly when they are stressed.[4] Newly introduced animals should be isolated for 1 month, and fecal culture should be considered.[4] Bleach is an effective disinfectant to use during an outbreak. Identification of carrier animals by fecal culture is recommended for herd problems. Vaccine efficacy is questionable, and to date its effects have not been thoroughly evaluated in sheep and goats.

Giardia: *Giardia*-induced diarrhea is more commonly seen in but not limited to 2- to 4-week-old lambs and kids.[4,30] The diarrhea is usually transient, but infected animals can continue to shed cysts for many weeks, even when they are clinically normal.[22,31,32] Therefore simply finding the agent in feces does not mean it is the cause of diarrhea, especially in older animals. However, these animals may be a source of infection for other animals and possibly humans.[22,30] Iodine-stained wet mounts of feces or tissue is the classic method of diagnosing giardiasis, but more sensitive immunologic techniques are now available.[22,30] Infected animals can be treated effectively with fenbendazole (5 to 10 mg/kg BID for 3 days or SID for 5 days).[22] *Giardia* has historically been treated with metronidazole (50 mg/kg PO SID for 5 days). However, use of this drug class in food animals is currently illegal in the United States. This is potentially a zoonotic condition.

Nutritional Diarrhea

Infectious agents are not the only cause of diarrhea in neonates. Nutritional problems can result in diarrhea, but these causes are overshadowed in the literature because the resulting diarrhea is usually mild and subsides without treatment. Nutritional diarrhea is most common in orphaned animals as a result of keepers offering poor-quality milk replacers, making mixing errors, or feeding large amounts infrequently (see Chapter 2). Diarrhea re-

sulting from consumption of lush pasture or high-energy rations is a common occurrence. In most cases such diarrhea is self-limiting. The incidence of this form of gastric upset can be minimized by a slow introduction (over 2 to 3 weeks) to energy-dense diets.

Calves with infectious diarrhea that develop maldigestion or malabsorption can have secondary nutritional diarrhea from an inability to digest carbohydrates (lactose, xylose).[33,34] This has been reported in goats, and also is probably a cause of diarrhea in lambs.[35] Diarrhea resulting from primary lactose deficiency also has been reported in calves.[36] Calves on poor-quality milk replacers can develop an overgrowth of normal enteric *E. coli,* resulting in diarrhea.[37] If lactose intolerance is suspected, decreasing the amount of lactose fed and using commercially available lactose enzymes may alleviate signs.

CAUSES OF DIARRHEA IN OLDER LAMBS AND KIDS

The most common cause of diarrhea in older lambs and kids is nematode infestation. This condition is discussed later in this chapter in the section on causes of adult diarrhea. Other major causes of diarrhea in older lambs and kids are *C. perfringens* and coccidiosis.

Clostridium perfringens

C. perfringens types A, B, C, and D can all cause diarrhea in lambs and kids, but type D is the most common agent.[4,7,38]

Pathogenesis. The disease occurs in peracute, acute, and chronic forms and is commonly called *enterotoxemia* or *overeating disease.* In the case of type C infection, a beta-toxin can cause acute hemorrhagic enteritis. Type C infection is seen mostly in lambs or kids younger than 3 weeks of age. An epsilon-toxin is responsible for pathology in type D infections. Enterotoxemia is usually seen in rapidly growing feedlot lambs on high concentrate rations. It also is associated with other feeding changes, including changes in type of pasture. However, it occasionally occurs with no reported dietary changes, particularly in goats.[4,7,39] This disease usually occurs in the fastest-growing and most well-conditioned animals. It can occur in vaccinated herds (again, more commonly in goats) so it should not be ruled out if a history of previous vaccination is present.[4]

Clinical signs. The peracute form of clostridial infection is characterized by the rapid onset of severe depression; abdominal pain; profuse, bloody diarrhea; and neurologic signs. Death occurs within hours of the onset of signs. Sudden death may occur without signs of diarrhea. The onset of neurologic signs followed by sudden death is more common in sheep, whereas goats are more likely to show signs of diarrhea before death.[4] Similar but less severe signs are seen in the acute form of the disease. The chronic form occurs more commonly in goats.[4,39]

Diagnosis. Antemortem diagnosis is based on clinical signs. At necropsy, *C. perfringens* can be cultured from intestinal tissue samples. However, the significance of a positive culture can be difficult to interpret because these organisms can be present in the gut normally and then proliferate after death. Histologic examination of sections of the gut can be helpful. Identification of the toxins (namely the epsilon-toxin) in intestinal contents is required for a definitive diagnosis.[4,7] Because the toxin degrades within several hours of death, not finding the toxin does not preclude enterotoxemia as a diagnosis.[38]

Treatment. Treatment is rarely effective but consists mainly of aggressive supportive care. *C. perfringens* type D antitoxins (15 to 20 ml SC) can be administered to animals during an outbreak of enterotoxemia if clinical signs are noted before death. The antitoxin may be more effectively used as a preventive in the face of an outbreak. During an outbreak any animals that have not been vaccinated should be given the antitoxin and vaccinated with the toxoid simultaneously; those previously vaccinated should receive a booster vaccination.

Prevention. Routine vaccination should start at 4 to 6 weeks of age and be followed by a booster 3 to 4 weeks later. However, on farms where the disease has become endemic, lambs or kids can be vaccinated and given antitoxin during the first week of life. Yearly vaccination, preferably a few weeks before the ewes and dams give birth increases colostral immunity in neonates and improves prevention programs. Goats may not respond as well to vaccination as sheep, so biannual or triannual vaccination is recommended, especially in problem herds.[4,38] Vaccination with only *C. perfringens* types C and D and tetanus is superior to the use of more polyvalent clostridial vaccines.[4] Reducing the energy density of the diet and avoiding sudden dietary changes or alterations of the feeding routine are crucial to prevention. Reducing internal parasites, particularly tapeworms, may further reduce the incidence of these disorders.

Coccidiosis

Pathogenesis. Coccidiosis is a protozoan parasitic disease that is a common cause of diarrhea in lambs and kids. It also may cause subclinical production losses.[19] Clinical disease is often seen when some form of stress (e.g., dietary change, weather changes, parturition, weaning) is occurring on the farm or in the flock. *Eimeria* species cause the disease in sheep and goats; each is infested with its own host-specific species. Unlike *Cryptosporidium,* which can be shed in feces in the infective stage, coccidia must sporulate outside the host to become

infective. Sporulation occurs under moderate temperatures and high moisture conditions. The nonsporulated and sporulated oocysts can survive a wide range of temperatures and may survive for years under certain conditions.

Clinical signs. Lambs and kids are most susceptible to the problem at approximately 1 to 4 months of age, although younger animals may become infected. Clinical disease is common after the stress of weaning, feed changes, or shipping. Crowded conditions result in excessive manure and urine contamination, which is ideal for the buildup and sporulation of the oocysts. Under these conditions, animals may be exposed to high numbers of infective organisms and develop diarrhea. The diarrhea in lambs and kids is *usually* not bloody, but it can contain blood or mucus and be very watery. Anorexia, dehydration, weakness, rough hair coat, and death all may occur.[19] Weight loss is common, and constant straining can result in rectal prolapse. In severe cases the disease becomes protracted because of necrosis of the mucosal lining. Even if these animals are treated appropriately, the diarrhea continues until the intestinal mucosa heals, which can take several days to weeks. Permanent scarring can result in chronic poor development, even if the diarrhea subsides.[19,40-42]

Diagnosis. Acute coccidiosis can be easily diagnosed from a direct smear or flotation of feces (Figure 4-10). In the chronic stages, most of the organisms have been shed and very low numbers are seen on fecal examination. Because normal animals can shed small numbers of pathogenic species or large numbers of nonpathogenic species, interpretation of fecal examinations in the chronic stages of coccidiosis or in animals with diarrhea from other causes can be difficult.[40-42] In these cases the clinician should rule out other diseases before making a diagnosis of coccidiosis. Blood analysis may show both anemia and hypoproteinemia.

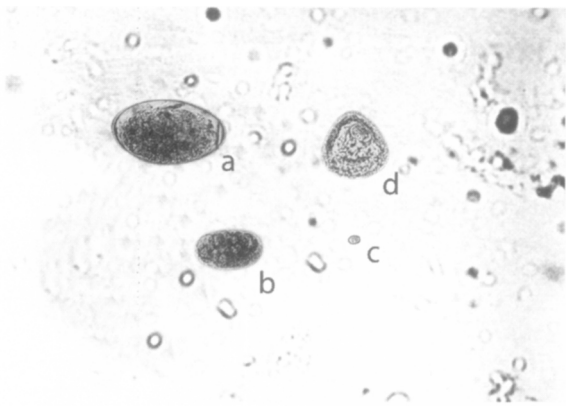

Figure 4-10 Parasites found on fecal examination. Trichostrongylid-type eggs (HOTC complex) *(b), Coccidia (c),* and tapeworm "eggs" *(d)* may be identified with flotation techniques. *Fasciola hepatica (a)* eggs are found with sedimentation techniques.

Treatment. Treatment of affected animals with clinical signs includes supportive care and administration of coccidiostats. All animals in the group should be treated during an outbreak. The use of coccidiostats has little effect on the existing infection, but it does prevent the spread of the disease from continued exposure to infective organisms.[40] Many coccidiostats inhibit coccidia development and prevent disease if given prophylactically.[40] They are of little value if they are given after the onset of clinical disease. Sulfa drugs appear to be clinically beneficial, but they may simply decrease secondary or concurrent bacteria-induced diarrhea.[40] Because coccidia develop some resistance to coccidiostats, these drugs should be administered only before stressful events (e.g., shipping, weaning, parturition).[40] The drugs listed in Table 4-3 and trimethoprim sulfa (15 mg/kg orally SID for 5 days) are approved for use in the United States.[7,41]

Prevention. Control involves improved sanitation and possibly the use of coccidiostats. Preventing overcrowding decreases the buildup of manure and infective oocysts. Exposure to sunlight and desiccation are two of the most effective means of killing the organisms. Minimizing stress and optimizing nutritional intake also are important. Coccidiostats available in the United States are shown in Table 4-3 and Appendix I. To avoid toxicity in growing animals, the clinician or keeper must carefully adjust dosages to the changing levels of feed intake as animals grow. All agents except amprolium should be fed for at least 4 weeks.[19,40] This allows exposure and subsequent development of immunity to occur while preventing the detrimental effects of clinical disease. However, coccidia can become resistant to coccidiostats; fecal samples should be periodically evaluated after prolonged use of a particular product. Anecdotal reports suggest amprolium resistance may occur on some farms. Moreover, if amprolium is offered with a creep feed rich in thiamine, its ability to act as a thiamine antagonist may be compromised. Year-round use of coccidiostats increases the potential for resistance. Therefore they should be fed only during times of expected risk.[40] The inclusion of lasalocid (1 kg of 6% premix) or decoquinate (1 kg of 13% premix) in 22 kg of trace mineralized salt fed as the only source of salt for 30 days prepartum can reduce the number of oocysts shed in ewe or dam feces. This practice can reduce the coccidia contamination of pasture and thereby remove a source of infection for kids and lambs. The benefits of administering lasalocid and monensin beyond coccidia control include increased feed efficiency, enhanced growth rate, and decreased incidence of free gas bloat. However, if coccidiostats are included in either mineral or feed supplements, inconsistent or depressed intake may result in subtherapeutic drug dosing.

Lambs are resistant to infection in the first few weeks of life. Exposure to the protozoa during this time confers immunity and resistance to later infections.[40,42]

TABLE 4-3

COCCIDIOSTATS USED FOR TREATMENT AND PREVENTION OF COCCIDIOSIS IN SMALL RUMINANTS*

COCCIDIOSTAT	DOSE	COMMENTS
Lasalocid	20 to 30 g per ton of feed; 0.5 to 1 mg/kg body weight per head per day in feed or salt	Approved for use in sheep in the United States
Decoquinate	0.5 mg/kg body weight per head per day in feed or salt	Approved for use in goats in the United States
Monensin	10 to 30 g per ton of feed	Approved for use in goats in the United States; may be most effective choice for goats
Amprolium	50 mg/kg body weight per head per day for 21 days (NOTE: This dose is five times the recommended calf dose)	Not approved for use in small ruminants in the United States; comes in liquid and crumble form; can potentially cause polioencephalomalacia at high doses and with prolonged administration
Sulfaquinoxaline	13 mg/kg body weight per head per day as 0.015% solution in water for 3 to 5 days	Approved for use in sheep in the United States
Sulfamethazine	119 to 238 mg/kg body weight per head per day in sheep; 50 g per ton of feed in goats	Not approved for use in small ruminants in the United States
Salinomycin	382 g per ton of feed in goats	Not approved for use in small ruminants in the United States

From Foreyt WJ: Coccidiosis and cryptosporidiosis in sheep and goats, *Vet Clin North Am: Food Anim Pract* 6(3):655, 1990; Craig TM: Coccidiosis in small ruminants, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV. Smith MC: Parasitic diseases of goats, *Proceedings of the 1996 Symposium on the Health and Disease of Small Ruminants,* 1996, Nashville, TN.
*Not approved for dairy animals in the United States.

MISCELLANEOUS CAUSES OF DIARRHEA IN KIDS AND LAMBS

Adenovirus, caprine herpesvirus, coronavirus, *Campylobacter jejuni, Yersinia* species, and *Strongyloides papillosus* can cause diarrhea in lambs and kids of various ages.[2,4,6] Enterohemorrhagic *E. coli* (EHEC) and enteropathogenic *E. coli* (EPEC) also have been isolated in the feces of kids with diarrhea.[43,44] These *E. coli* types are K99- and F41-negative. Culture and serotyping of these organisms from feces and tissue samples with typical histopathologic lesions is diagnostic. Although ETEC is not zoonotic, EHEC and EPEC can potentially affect humans.

TREATMENT OF LAMBS AND KIDS WITH DIARRHEA

Although some causes of diarrhea have specific treatments, many animals need to be treated for dehydration and metabolic acidosis regardless of the inciting cause. Animals with only mild diarrhea, especially mild nutritional diarrhea, may not require therapy unless they become dehydrated. If kids or lambs become less than 8% dehydrated and are only mildly depressed but still willing to nurse, they can be treated with oral electrolytes designed for calves. Fluids can be administered by bottle or by tube if the animal will not nurse. The keeper or clinician should carefully adjust the amount of fluids for lambs and kids (250 to 500 ml, or 8 to 16 oz, as opposed to 4 L in a calf). Because most electrolyte solutions designed for calves contain glucose, after they have been mixed they should be refrigerated and any leftovers discarded within 24 hours. IV fluids may be needed to treat more severe dehydration. If the lamb or kid is too weak to stand, IV fluids are indicated. Isotonic fluids containing electrolytes should be given to replenish losses. Glucose can be added to fluids to make a 1% to 2.5% solution. Sodium bicarbonate also may be administered, especially if the dehydration is severe. A rule of thumb is to give one fourth of the calculated fluid need (see Appendix II) as isotonic bicarbonate (1.3%). Extra potassium (10 to 20 mEq/L) can be added to fluids because most animals are severely dehydrated from diarrhea and depleted in potassium, even though their blood potassium levels may be elevated. If extra potassium is added, acidosis must be corrected concurrently. After correcting the dehydration, the keeper or clinician can offer oral electrolyte-enriched fluids to replace ongoing losses caused by continued diarrhea.

Removing milk or milk replacer from the diet is not recommended. Young animals need nutrients, and even high-energy, glucose-containing electrolyte solutions are

no substitute for milk. Animals should continue to receive milk replacer in normal amounts or be allowed to nurse; they can be supplemented with oral electrolytes if necessary. Animals being hand fed should be offered small amounts frequently to help minimize problems. Electrolytes should never be mixed with milk, but should instead be given in separate feedings. If lactose deficiency is suspected, lactase drops or capsules (available in health food stores) can be added to milk or milk replacer.[35]

NSAIDs (flunixin meglumine, 1.1 to 2.2 mg/kg IV; ketoprofen, 2.2 to 4.4 mg/kg IV) are beneficial, especially if toxemia is involved, as in ETEC, enterotoxemia, and salmonellosis. It is the authors' opinion that antimicrobial agents should be reserved for proven outbreaks of salmonellosis and for animals with other causes of diarrhea that do not respond to fluid therapy and NSAIDs; these drugs should only be administered parenterally. The use of oral coating agents and antacids is popular, but it has not been shown to be beneficial and is not therapeutically logical in light of the pathogenesis of these diseases. Probiotics may be beneficial in reestablishing the normal flora of the small intestine. The authors' rule of thumb is that nothing should be given orally except milk, oral electrolytes, and probiotics.

GENERAL CONTROL MEASURES FOR INFECTIOUS DIARRHEA

Ensuring adequate intake of high-quality colostrum and minimizing stress are important for prevention of all neonatal diseases. A normal lamb or kid will stand and nurse within 45 minutes to 1 hour of birth. The ingestion of colostrum within 2 to 3 hours is essential in preventing hypothermia and hypoglycemia and decreasing the incidence of various diseases. Lambs or kids born as twins or triplets, weak or injured neonates, those born during severe weather, those born from a dam with dystocia, and those delivered by Cesarean section are all candidates for colostrum supplementation. If supplemental colostrum is provided, it should be good-quality colostrum from females that have tested negative for Johne's disease, ovine progressive pneumonia (OPP), and caprine arthritis-encephalitis (CAE). Mixing colostrum from several cows decreases the incidence of the "cow colostrum–associated" hemolytic disease sometimes seen in lambs. If the lamb or kid is unable to nurse, it should be tube fed 50 ml/kg of colostrum. The veterinarian or animal handler can sit comfortably holding the lamb or kid in sternal recumbency in the lap. A 12 to 14 French soft feeding tube is then lubricated, inserted into the side of the mouth, and passed slowly. If the tube is placed in the trachea, the lamb or kid will become uncomfortable and may shake and cough. The tube may be palpated on the left side of the throat. After the tube has been slowly passed to the thoracic inlet, colostrum can be administered by gravity flow (see Chapter 6).

Prepartum shearing of the dam may decrease the ingestion of feces by lambs. Good sanitation of lambing and kidding areas is paramount in management programs that stress prevention. The presence of organic matter interferes with the effectiveness of many disinfectants, so removal and proper disposal of feces, carcasses, and placentas are essential. When disposing of waste material containing either *Cryptosporidium* or *Giardia,* the keeper should be careful to avoid contaminating water sources. Infected animals should be isolated to prevent spread of the infection throughout the flock. In general, infected animals should remain in the environment where the infection was first diagnosed, because it is already contaminated. Removing pregnant ewes or dams to a clean area before lambing or kidding helps minimize the continued spread of disease. If possible, lambs and kids already born but not showing clinical signs should be removed to a third area. If "safe" pastures are maintained for internal nematode control, they are ideal for use in an emergency situation to control these diseases. Although some animals may appear normal, they may be incubating and possibly shedding the infective agents of a disease. If such animals are moved with pregnant females, they can be a source of contamination in a clean area. If healthy lambs and kids cannot be moved to a third, relatively safe area, they should be left with the clinically infected animals because they have already been exposed.

REFERENCES

1. Sherman DM: Causes of kid morbidity and mortality: an overview, *Proceedings of the Fourth International Conference on Goats,* Brasilia, Brazil, 1987, EMBRAPA-DDT.
2. Vickers MC: Enteric infections in young goats and their control, *Proceedings of the Sixteenth Seminar, Sheep and Beef Cattle Society,* Palmerston North, New Zealand, 1986.
3. Blackwell TE: Enteritis and diarrhea, *Vet Clin North Am: Large Anim Pract* 5(3):557, 1983.
4. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
5. Nagy B et al: Infectious gastrointestinal diseases of young goats, *Proceedings of the Fourth International Conference on Goats,* Brasilia, Brazil, 1987, EMBRAPA-DDT.
6. Nagy B et al: Occurrence of cryptosporidia, rotaviruses, coronavirus-like particles and $K99^+$ *Escherichia coli* in goat kids and lambs, *Proceedings of the Third International Symposium on Veterinary Laboratory Diagnostics,* Ames, Iowa, 1983.
7. Kimberling CV: *Jensen and Swift's diseases of sheep,* ed 3, Philadelphia, 1988, Lea & Febiger.
8. Hodgson JC: Escherichia coli *in domestic animals and humans,* Wallingford, UK, 1994, Cab International.
9. Runnels PL, Moon HW, Schneider RA: Development of resistance with host age to adhesion of $K99^+$ *Escherichia coli* to isolated intestinal epithelial cells, *Infect Immun* 28(1):298, 1980.
10. Zeman DH, Thomson JU, Francis DH: Diagnosis, treatment, and management of enteric colibacillosis, *Vet Med* 84(8):794, 1989.

11. Schultheiss P: Diarrheal disease in calves, *Large Anim Vet* 47(2):24, 1992.
12. Roussel AJ et al: Effect of flunixin meglumine on *Escherichia coli* heat stable enterotoxin-induced diarrhea in calves, *Am J Vet Res* 49(8):1431, 1988.
13. Theil KW et al: Group B rotavirus associated with an outbreak of neonatal lamb diarrhea, *J Vet Diagn Invest* 7(1):148, 1995.
14. Babiuk LA, Sabara M, Hudson GR: Rotavirus and coronavirus infections in animals. In Karger S, editor: *Infection and immunity in farm animals,* Switzerland, 1985, Basel.
15. Theil KW, Lance SE, McCloskey CM: Rotaviruses associated with neonatal lamb diarrhea in two Wyoming shed-lambing operations, *J Vet Diagn Invest* 8(2):245, 1996.
16. Munoz M et al: Rotavirus excretion by kids in a naturally infected goat herd, *Sm Rum Res* 14(1):83, 1994.
17. Heath SE: Neonatal diarrhea in calves: diagnosis and intervention in problem herds, *Comp Cont Ed Pract Vet* 14(7):995, 1992.
18. Moore JA, Blagburn BL, Lindsay DS: Cryptosporidiosis in animals including humans, *Comp Cont Ed Pract Vet* 10(3):275, 1988.
19. Foreyt WJ: Coccidiosis and cryptosporidiosis in sheep and goats, *Vet Clin North Am: Food Anim Pract* 6(3):655, 1990.
20. Berg IE, Peterson AC, Freeman TP: Ovine cryptosporidiosis, *J Am Vet Med Assoc* 173(12):1586, 1978.
21. Sanford SE et al: Cryptosporidiosis, rotaviral, and combined cryptosporidial and rotaviral infections in goat kids, *Can Vet J* 32(10):626, 1991.
22. Rings DM, Rings MB: Managing *Cryptosporidium* and *Giardia* infections in domestic ruminants, *Vet Med* 91(12):1125, 1996.
23. Corwin RM: Cryptosporidiosis: a coccidiosis of calves, *Comp Cont Ed Pract Vet* 14(7):1005, 1992.
24. Webster KA et al: Detection of *Cryptosporidium parvum* oocysts in feces: comparison of conventional coproscopical methods and the polymerase chain reaction, *Vet Parasit* 61(1/2):5, 1996.
25. Campbell I et al: Effect of disinfectants on survival of *Cryptosporidium* oocysts, *Vet Rec* 111(18):414, 1982.
26. Perryman LE et al: Protection of calves against cryptosporidiosis with immune bovine colostrum induced by a *Cryptosporidium parvum* recombinant protein, *Vaccine* 17(17):2142, 1999.
27. Bulgin MS, Anderson BC: Salmonellosis in goats, *J Am Vet Med Assoc* 178(7):720, 1981.
28. House JD, Smith BP: Current strategies for managing salmonella infections in cattle, *Vet Med* 93:756, 1998.
29. Cohen ND et al: Comparison of polymerase chain reaction and microbiological culture for detection of salmonella in equine feces and environmental samples, *Am J Vet Res* 57:780, 1996.
30. Kirkpatrick CE: Giardiasis in large animals, *Comp Cont Ed Pract Vet* 11(1):80, 1989.
31. Koudela B, Vitovec J: Experimental giardiasis in goat kids, *Vet Parasit* 74(1):9, 1998.
32. Olsen ME et al: Effects of giardiasis on production in a domestic ruminant (lamb) model, *Am J Vet Res* 56(1):1470, 1995.
33. Nappert G et al: Determination of lactose and xylose malabsorption in preruminant diarrhetic calves, *Can J Vet Res* 57(8):152, 1993.
34. Naylor JM: Neonatal ruminant diarrhea. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
35. Weese JS, Kenney DG, O'Connor A: Secondary lactose intolerance in a neonatal goat, *J Am Vet Med Assoc* 217(3):372, 2000.
36. Olchowy TWJ et al: Lactose intolerance in a calf, *J Vet Int Med* 7(1):12, 1993.
37. Roy JHB: *The calf,* ed 4, London, 1980, Butterworth.
38. Uzal FA, Kelly WR: Enterotoxemia in goats, *Vet Res Commun* 20:481, 1996.
39. Songer JG: Clostridial diseases of small ruminants, *Vet Res* 29:219, 1998.
40. Craig TM: Coccidiosis in small ruminants, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
41. Smith MC: Parasitic diseases of goats, *Proceedings of the 1996 Symposium on the Health and Disease of Small Ruminants,* 1996, Nashville, TN.
42. Clarkson MJ, Winter AC: *A handbook for the sheep clinician,* ed 5, Liverpool, UK, 1997, University Press.
43. Duhamel GE, Moxley RA, Maddox CW: Enteric infection of a goat with enterohemorrhagic *Escherichia coli* (0103:H2), *J Vet Diagn Invest* 4(2):197, 1992.
44. Drolet R, Fairbrother JM, Vaillancourt D: Attaching and effacing *Escherichia coli* in a goat with diarrhea, *Can Vet J* 35(2):122, 1994.

DIARRHEA IN ADULT SHEEP AND GOATS

The differential diagnosis list for acute and chronic diarrhea in small ruminants is very long.[1] The most common cause of diarrhea in adult sheep and goats is parasitism; another major cause is Johne's disease. Both of these diseases are discussed in the following sections. Other causes of acute diarrhea include rumen acidosis, peritonitis, endotoxemia, and ingestion of toxins. The list of toxins that cause diarrhea also is very long, and often the diarrhea is not the primary clinical sign. Some of the more common toxins that produce diarrhea are arsenic, toxic amounts of salt, levamisole, copper, oak, selenium, and pyrrolizidine alkaloids.[1] *Salmonella* species and chronic enterotoxemia can cause diarrhea in adult animals. Coccidiosis can occur in adults under severe stress or in animals that possess limited immunity because of lack of exposure. Hepatic and renal disease and copper deficiency are sometimes accompanied by chronic diarrhea, but weight loss is a more common sign in adults.

INTESTINAL PARASITISM

Nematode Infestation

Etiology and pathogenesis. Sheep and goats are infested with many of the same gastrointestinal nematode parasites as cattle, but these parasites tend to either be species-specific or have some amount of host specificity. Sheep and goats are susceptible to the same nematodes and tend to share resistance to those that infect cattle and horses. The major gastrointestinal nematodes that parasitize pastured sheep and goats are *Haemonchus, Ostertagia, Trichostrongylus, Cooperia, Nematodirus, Oesophagostomum,* and *Bunostomum* species. The acronym *HOTC* comes from the first letter of each of the first four genera of parasites listed. The specific parasites that produce

disease vary from flock to flock. Climate usually determines which parasites are of clinical significance on a farm, and the weather determines when the parasites will be transmitted and infective. In much of the United States, *Haemonchus* is the most significant parasite with respect to both clinical disease and anthelmintic resistance. Most of these parasites affect the abomasum or small intestine of young, recently weaned animals and occasionally adult animals. Sheep (and, to a lesser extent, goats) that are older than 18 months may be less susceptible. Overcrowding and overgrazing with concurrent pasture mismanagement and malnutrition usually increase susceptibility to these parasites. Inadequate nutrient or protein intake may result in greater susceptibility.[2]

The life cycle appears to be similar in most of these parasite species. Adults lay eggs that are passed in the feces; except for *Nematodirus* species, the eggs hatch under favorable environmental conditions. The larvae go through several free-living developmental stages becoming infective. When the infective larvae are ingested by the host, the parasite completes its life cycle as an adult. *Trichuris* eggs are the infective stage and can survive for extended periods in dry lots or barns.[4] However, *Trichuris* is associated with minimal pathology. During dry environmental conditions, fecal pellets tend to trap the nematode larvae, whereas in wet conditions, larvae are released onto the pasture. Therefore drought conditions followed by rain can result in devastatingly high rates of pasture contamination as larvae that have remained in fecal pellets are released.[2]

Very high environmental temperatures result in shorter survivability of some stages of infective larvae. Most of the larvae have adapted the ability to over-winter, but can survive only for short periods outside the host during spring. *Nematodirus* is an exception in that the developmental stages leading to infective larvae occur while the microbe is still encapsulated in the egg. However, compared with other species of parasite, *Nematodirus* is of minor importance. *Nematodirus battus* may pose a threat to young, newly weaned and therefore immunologically naive grazing lambs. The hookworm *Bunostomum* also is different, as it may infect the host by either oral ingestion or percutaneous penetration. With the exception of the small intestinal parasite *Strongyloides,* lambs or kids fed indoors or in dry pens tend to be free of parasites.

Clinical signs. All intestinal nematode infections produce similar signs, although infection with the more rarely encountered *Bunostomum* may perhaps result in more profound anemia. If they infest the animal in sufficient numbers, all nematodes may cause poor growth, decreased feed conversion, decreased milk production, weight loss, diarrhea, anemia, ventral edema (bottle jaw), midline edema, and death. Again, all these parasites can potentially result in disease, but *Haemonchus* is the most devastating, particularly in more temperate regions.[2]

Diagnosis. Antemortem diagnosis of nematode infestation is made by examining the feces for nematode eggs. Although a direct fecal smear can be examined, the mere presence of parasite eggs is not helpful in determining the parasite load of an animal or animals. Quantifying of the EPG of feces is the best way of estimating parasite loads. The quantitative McMaster's technique for determining the EPG of feces is shown in Box 4-1. Common nematode eggs are shown in Figure 4-10.

Treatment and control program. After taking a thorough history of the previous parasite control program used on a farm and determining its effectiveness, the clinician can design and implement a new control program.[2,3] However, before deciding to implement a deworming program, the clinician should decide which parasites are in need of control and whether control of these parasites is cost effective in a particular flock. Whenever possible, a dewormer that can reduce EPG counts on the farm by 90% should be identified and used for at least a year. Of all the parasite prevention programs, strategic deworming or a combination of strategic and tactical programs appear to produce the best results.[2,4,5]

Strategic deworming is used when most of the parasites are inside the animals and not on the pasture.[2] In northern climates, strategic deworming can best be carried out during the winter, when the nematode parasites are in a hypobiotic state. When environmental conditions are inhospitable for the survival of the infective larvae, some of the most pathogenic nematodes (e.g., *Haemonchus)* may become hypobiotic; that is, they assume a state of arrested development. They may then mature to the adult stage when environmental conditions become conducive for the survival of their eggs or larvae. Preventing or decreasing the numbers of maturing adults by killing the larvae before the periparturient rise in parasite egg production and pasture contamination is an excellent management tool.[4,6,7] Unfortunately, in warmer, more temperate to subtropical environments, this method is less effective because larvae can survive the environment for longer periods. The addition of a protein supplement overlapping the expected periparturient rise has been shown to decrease the number of parasite eggs shed around the time of parturition. However, the cost of the protein supplement may outweigh its benefits.[8]

A strategic program entails the use of an anthelmintic agent that is capable of killing encysted larvae. Animals are then moved to parasite-free or safe pastures—areas in which the level of parasite contamination is too low to result in infection of grazing animals. Examples of safe pastures include pastures where sheep or goats have not grazed for 3 to 6 months in the spring or fall, respectively (and depending on the climate); pastures used for hay production; new pastures (i.e., those used for corn, cotton, or other crops); and pastures grazed by horses or cattle. The use of safe pastures is paramount in any de-

worming program. Rotating pastures after less than 3 months during the warm part of the year or less than 6 months during cooler months is ineffective.[3] However, if pastures are tilled and replanted, by the time new grazeable forage is available, infective parasite larvae will be dead or significantly decreased.[3]

An alternative to pasture rotation is to perform an initial strategic deworming before lambing or kidding and follow it with two to four more dewormings at 3-week intervals throughout the lambing and kidding period.[9-11] Treating lambs or kids at weaning and moving them to a safe pasture is a form of strategic deworming.[4] In lambs or kids to be sold at an early age, the administration of a single anthelmintic treatment followed by a move to a safe pasture may be all that is required. A "double treat and move" system is required for lambs kept for 12 to 18 weeks after weaning, particularly during the summer.[12,13] This form of strategic deworming requires two treatments 6 to 8 weeks apart as well as two safe pastures. In northern climates where animals are moved to a dry lot or barn for the winter, a strategic anthelmintic administration as animals are moved off pasture can help reduce the parasite burden through the winter.[14] If this deworming is followed by minimal or no exposure to grazing areas and another dewormer is administered before the spring rise in fecal egg counts, the total parasite burden on spring pasture can be drastically reduced, effectively controlling parasites until summer or fall.

Tactical deworming programs are used to remove parasites from their hosts before they enter their reproductive phase and can contaminate the pasture.[4] An example of tactical deworming is treating animals 10 to 14 days after a rain, particularly if the rain has followed a drought. Parasite transmission is worse in most flocks during this time as pastures become heavily contaminated. McMaster's counts of more than 1000 EPG in the spring or more than 2000 EPG in the fall warrant tactical deworming.[2,4,7]

Opportunistic deworming and salvage deworming are usually less effective in long-term flock management. Many times salvage deworming programs are used to save the lives of heavily parasitized animals.[4] If animals are dewormed only after showing signs of parasitism (e.g., bottle jaw, anemia), animal and flock productivity have already been depressed. Deworming during handling for other procedures (e.g., castration, vaccination, shearing) is an example of an opportunistic program. It is convenient but is not conducive to long-term flock health. Flock work should be scheduled around parasite management programs, not vice versa.[2,14]

Suppressive deworming programs entail the use of anthelmintics at regular intervals, usually every 2 to 4 weeks. Suppressive programs are labor-intensive, tend to be very expensive, fail to identify animals with superior immunity to parasites, and ultimately result in anthelmintic resistance despite initial effectiveness.[2,4]

As a general rule, the more frequently deworming occurs, the more quickly resistance is attained to anthelmintics. After deworming, only resistant parasites remain to infect the animal and they are able to reproduce freely, resulting in proliferation of resistant strains.[2,4] Using drugs that remain in tissues at inappropriately low concentrations and treating and retaining immunocompromised animals encourage the development of anthelmintic resistance. Practices that ensure adequate dosages, proper treatment techniques, and appropriate types of anthelmintics should be emphasized.[2,13]

The clinician should do everything in his or her power to minimize the incidence of anthelmintic resistance, both through their own actions and by counseling owners in proper use of deworming drugs. The product development market for anthelmintics is the cattle industry. The small sheep and goat markets simply use drugs made available for cattle. Because most available anthelmintics are highly effective in controlling parasites, anthelmintic resistance in sheep and goats must be avoided. The anthelmintics that have been used previously on a flock, the route of administration (e.g., PO, SC, IM, pour-on), and the length of use should be determined. Few dewormers are approved for use in sheep and goats, but many approved for use in cattle and horses may be effective.[2,7] If sheep graze with goats that harbor anthelmintic-resistant parasites, the sheep also may become infected.[4] However, if sheep are allowed to graze while the goats browse and the two groups rarely mingle, less parasite movement will occur between these species.

Resistance to macrolides (e.g., ivermectin, doramectin, moxidectin) does occur. Resistant worms are generally not very tolerant of cold temperatures and therefore resistance to this drug class in northern environments is not as large a problem as it is in more temperate or subtropical zones.[15] Although moxidectin is not approved for use in sheep and goats in the United States, it has been shown to be effective in cases where ivermectin resistance is encountered.[2] Still, this drug should be avoided until all other anthelmintics have failed. Craig[3,4] has suggested that clinicians refrain from injecting or using pour-on macrolide preparations designed for cattle in small ruminants. This practice may enhance the development of resistant strains of some internal parasites because of inappropriately low drug absorption (with pour-on use) or long-term subtherapeutic levels (with injection).[4]

If resistance to tetrahydropyrimidines (e.g., morantel, pyrantel) occurs in a flock, levamisole also may be ineffective. Morantel and levamisole resistance in parasites appears to be sex-linked. Therefore if animals are not exposed to these drugs for several years, reversion to susceptibility can occur.[3,4]

If resistance to one of the benzimidazole dewormers has been documented in a flock, some resistance to all members of that class is likely.[4] Benzimidazole-resistant *Haemonchus* species appear to be more virulent, produce more eggs, cause greater environmental contamination,

and survive in the environment as free-living larvae for longer periods. Benzimidazole-resistant parasites apparently do not revert to susceptible forms, even over long periods. Therefore the clinician or keeper should exercise caution to minimize resistance.[4] Benzimidazole efficacy can be improved by increasing dosages, dividing dosages into two treatments administered at 12-hour intervals, and instituting pretreatment fasting.[2]

If resistance to numerous classes of anthelmintics occurs on a farm, combining two of the resistant classes of dewormers (fenbendazole and levamisole) has proven effective.[16] When using combined dewormers, the clinician should administer the full therapeutic dosage of each. Anthelmintics are metabolized at different rates by sheep and goats. Goats may require larger dosages of some dewormers than sheep.[7] Craig[4] has suggested that if no dose rate is known for a particular anthelmintic for sheep or goats, the animals should be treated at twice the suggested cow dosage. Pour-on anthelmintics designed for cattle tend to be of limited value when used topically on either goats or sheep.[2] A list of dewormers useful in sheep and goats is listed in Table 17-3. To maximize a parasite control program, anthelmintics that appear effective should be used for only 1 year before a new class of deworming drug is used. More frequent rotation (after less than 1 year) of anthelmintic agents hastens resistance and should be avoided whenever possible.

Whenever a flock is dewormed, animals should be treated based on the heaviest animal in the group and not on the group's average weight. Underdosing can hasten the formation of parasitic resistance and therefore should be avoided.[4] Holding the sheep or goats in a dry lot overnight or feeding only dry hay for 12 to 24 hours before and 12 hours after deworming appears to improve the efficacy of some orally administered anthelmintic agents (benzimidazole). Limiting feed intake before deworming slows the rate of passage of ingesta through the bowel, enhancing drug effectiveness.[2,13,14] Feed should never be withheld from sick or debilitated animals or late-term females.[2,13,14] Most dewormers may effectively control adult or larval parasites but are ineffective against eggs. Therefore animals should be kept on a dry lot for as long as 3 days after deworming, then moved to a safe pasture. Use of this procedure minimizes parasite egg contamination of the new pasture because most of the egg-contaminated feces is voided within 72 hours of deworming. If more than one dosage appears on the drug label, the larvacidal dose should be used (fenbendazole at 10 mg/kg rather than 5 mg/kg).[2]

Anthelmintic effectiveness can be determined by comparing a McMaster's fecal EPG on the day of deworming with one taken 7 to 14 days later. If less than a 90% drop in EPG is found, anthelmintic resistance exists and the animals should be switched to another class of dewormer.[2] Although it is a controversial method, the authors have used this technique to identify anthelmintic effectiveness for many years and on many farms and ranches.[2,14] The authors randomly collect feces from 5% to 10% (or a minimum of 10 animals) of the sheep or goats on the farm. A composite sample is prepared by combining equal amounts of stool from all animals. Craig[3] has suggested that combining stool samples from many animals alters the accuracy of the tests because great individual variation in fecal egg counts occurs among animals. Composite egg counts more accurately reflect parasite burdens in groups of young animals, and individual fecal examinations are more accurate in adults.[2,4] Still, the authors prefer to use composite samples unless obvious differences in stool character or body condition score exist among the sampled animals. Anthelmintic resistance can be minimized by using drugs that reduce fecal egg counts by 90%. Pre- and postdeworming changes in EPG should be evaluated yearly or whenever resistance is suspected.[4] In vitro methods of assessing flock parasite resistance also are available at some diagnostic laboratories. In most in vitro tests, larvae are hatched from collected feces and the sensitivity of different anthelmintics is determined by larval exposure. These tests are very accurate but tend to be quite expensive.

The most effective method to prevent anthelmintic resistance is to not use deworming drugs at all.[4] One of the most overlooked management procedures is the identification and selection of parasite-resistant sheep and goats.[2] Some breeds or familial lines within breeds have excellent parasite resistance (e.g., Gulf Coast Native and Barbados sheep, some strains of Spanish, Pygmy, and Tennessee myotonic-fainting goats).[2] One study[17] comparing Boer-crossed goats with non-Boer crosses found that the Boer crosses had significantly more parasite infestations. Only a small number of flock members contribute the greatest amount of environmental parasite contamination because susceptible animals shed the most eggs in their feces. Animals with the lowest EPG in a flock may be those that possess the most parasite resistance.[4] Salvage deworming programs should generally be avoided, but they may be used as aggressive selection criteria. That is, animals that do well with little or no deworming, particularly those grazing heavily contaminated pastures, should be identified and retained in the breeding flock. Those that become infected should be dewormed to salvage them or save their lives and then sold when possible. Proper record keeping and identification of all animals is paramount in selecting for parasite resistance.[2,14] This aggressive approach can yield excellent results if it is carefully implemented, but devastating losses can occur if it is poorly managed.

When introducing new animals to a flock, keepers should have biosecurity programs in place to limit the introduction of new or potentially anthelmintic-resistant parasites. New flock additions should be kept in a dry lot for 3 weeks and dewormed at least twice with two different classes of dewormers during this period. The effectiveness of the anthelmintic agent used should be deter-

mined by fecal examination before the animal is allowed contact with the rest of the flock.[2]

Other nontraditional chemical methods of parasite control are used by some owners. Some appear to be worthless (e.g., diatomaceous earth), but others (e.g., nematophagous fungi, herbal dewormers) may prove effective in some situations.

Cestode Infestation

Pathogenesis. The most common gastrointestinal tapeworm of sheep and goats seen in North America belongs to the genus *Moniezia.* Cestodes (tapeworms) are usually of more concern to owners than clinicians, who generally consider them only incidental low-grade pathogens, particularly in adult animals. Still, several 10- to 20-foot-long tapeworms can compete with the host for nutrients, hinder normal gut motility, and excrete some toxic wastes into the host's gastrointestinal tract. Mature tapeworm eggs are passed in the feces individually or protected in proglottides, which are usually visible to the owner. The eggs embryonate and infect a mite, a small pasture-living arthropod that serves as the intermediate host. A sheep or goat ingests the mite while grazing, allowing the tapeworm to complete its life cycle.

Clinical signs. Tapeworms may rarely cause disease in lambs and kids less than 6 months of age. Anecdotal reports suggest a cause-effect relationship between heavy tapeworm infestation and an increased incidence of *C. perfringens* enteritis, digestive disturbances (e.g., diarrhea, constipation), poor condition, and anemia. Ulceration at the site of attachment may be seen on necropsy. Rarely species of *Trypanosoma,* the fringed tapeworm, may cause liver condemnation.

Diagnosis and treatment. A presumptive diagnosis can be made by finding proglottides in the stool, eggs on direct smears, or eggs on fecal flotations (see Figure 4-10). Treatment with albendazole (15 mg/kg), fenbendazole (20 to 25 mg/kg), or praziquantel (10 to 15 mg/kg) may be effective either with a single treatment or with daily therapy (e.g., fenbendazole daily for 3 to 5 days). Because of the free-living nature of the arthropod intermediate host, animals are readily reinfected after treatment, which may give rise to the false assumption that the therapy was ineffective. Again, tapeworm infestation *may* result in disease, but often it is easier to blame the tapeworm segment seen in the stool as a cause of disease than to implicate the unseen thousands of HOTC complex parasites in the abomasum and small intestine of the animal.[3,14]

Johne's Disease

Johne's disease (also called *paratuberculosis*) is a chronic wasting and diarrheal disease caused by the bacteria *Mycobacterium avium* subspecies *paratuberculosis.* Transmission of the organism is primarily by the fecal-oral route. Young animals are more susceptible to infection than adults. It can be transmitted through milk and placenta.

Pathogenesis. Bacterial shedding in feces and milk and transplacental transmission is more common in animals showing clinical signs.[18-20] Therefore the offspring of infected animals and especially the offspring of animals showing clinical signs are most likely to acquire the infection. After an animal is exposed, it will either clear the organism or develop a chronic, persistent infection. The infection is most commonly isolated to the ileal regions of the small intestine, where it causes granulomatous thickening of the intestine and subsequent malabsorptive diarrhea. Infected animals may be asymptomatic for years.

Clinical signs. Morbidity rates are low (approximately 5%), but for every animal with clinical signs, several exist in the subclinical state, and may be a source of both horizontal and vertical transmission.[18] Both sheep and goats appear to remain asymptomatic until 2 to 7 years of age. The most consistent clinical sign in sheep and goats is chronic weight loss. Chronic diarrhea occurs in approximately 20% of cases.[18] Signs may appear with or be exacerbated by stress, especially after parturition.[18,19] Hypoproteinemia and chronic mild anemia are the only consistent clinicopathologic findings. Because of their low protein levels, infected animals can develop submandibular edema.

Diagnosis. Diagnosis is by culture of the organism from feces. Unfortunately, this testing takes between 8 and 14 weeks, but it can detect 40% to 60% of clinically infected goats. Sheep strains of Johne's disease and some goat variant strains seem to be more difficult to culture in media used to identify cattle strains of the disease. Therefore fecal culture in sheep and goats appears to be of limited benefit.[19,20] A relatively inexpensive and easily performed method of identifying approximately 50% of all clinically infected animals is acid-fast staining of fecal smears.[18,19] A PCR test of feces also is available, but its sensitivity is lower than that of fecal culture. Good diagnostic results can be obtained with serologic testing for antibodies (e.g., agar gel immunodiffusion [AGID], enzyme-linked immunospecific assay [ELISA], complement fixation) in animals showing clinical signs. The specificity of all the serologic tests is greater than 95% in sheep and goats with signs of clinical disease, although the sensitivity is not as high.[19-21] Therefore a positive serologic test in an animal showing clinical signs indicates that the animal has Johne's disease. However, the disease cannot be ruled out with a negative test. Sheep and goats appear to respond differently in regard to the formation of antibodies. Sheep tend to develop antibodies in the later stages of the disease, whereas antibodies may be detected much earlier in the

goat. The AGID test appears to be the best serologic test currently available.[22,23] The ELISA and complement fixation tests can cross-react with *Corynebacterium pseudotuberculosis,* making them of limited value in flocks with caseous lymphadenitis infections.[19,24] Necropsy diagnosis is based on the finding of thickened, corrugated intestines, especially in the area of the ileum. Acid-fast staining of impression smears (taken from the ileum and ileocecal lymph nodes) can help yield a quick diagnosis. The staining of numerous clumps of acid-fast rods is highly suggestive of Johne's disease.

Prevention. Johne's disease has no effective treatment, so prevention and control are imperative. However, preventing the introduction of Johne's disease into a herd can be difficult. Because animals with subclinical infection may not shed the organism or may shed only small quantities of it, fecal culture is helpful only if a positive culture is obtained. The sensitivity of serologic tests of animals with subclinical disease is low and variable among flocks.[19,20] Negative test results in subclinically infected animals are common. However, the specificity of serologic tests remains high, and therefore a positive test is a valid reason to not purchase an animal.[19] Because Johne's disease also occurs in cattle, supplemental colostrum supplies should come only from dairy herds with no history of Johne's disease.

After Johne's disease is diagnosed in a herd, several control measures can be taken. Sanitation is important because the organism is highly resistant in the environment (able to survive more than 1 year under most conditions).[20] Reduced stocking rates, frequent cleaning of pens, and use of automatic waterers decrease fecal transmission. Keepers should cull the offspring of infected animals. Culling animals based on the results of AGID tests or fecal culture of the flock is recommended. Animals should be tested at least once a year. More frequent testing as resources allow speeds the identification of infected animals. A vaccine for cattle is only available in some locales and clinicians or keepers may require official permission to use it. Vaccine use does not eliminate infection, but it can decrease herd prevalence, delay the onset of clinical signs, and decrease cross-transmission by infective bacterial shedding in the feces.

References

1. Smith BP: Alterations in alimentary and hepatic function. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
2. Pugh DG, Mobini SM, Hilton CD: Control programs for gastrointestinal nematodes in sheep and goats, *Comp Cont Ed Pract Vet* 20(4 suppl.):S112, 1998.
3. Craig TM: Epidemiology of internal parasites: effects of climate and host reproductive cycles on parasite survival, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
4. Craig TM: Anthelmintic resistance: the selection and successful breeding of superior parasites, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
5. Menzies P: Control and prevention of specific diseases of sheep and goats, *Proceedings of the Symposium on Health and Disease of Small Ruminants,* Nashville, TN, 1996, American Association of Small Ruminant Practitioners.
6. Herd RP: Control of periparturient rise in worm egg counts of lambing ewes, *J Am Vet Med Assoc* 182:375, 1983.
7. Bretzlaff K: Production medicine and health programs for goats. In Howard JL, editor: *Current veterinary therapy in food animal practice 2,* Philadelphia, 1986, WB Saunders.
8. Donaldson J: The effects of dietary protein on establishment and maturation of nematode populations in adult sheep. In Barrel GK, editor: *Sustainable control of internal parasites in ruminants,* Christchurch, UK, 1997, Lincoln University.
9. Herd RP: Nematode infections—cattle, sheep, goats, swine. In Howard JL, editor: *Current veterinary therapy in food animal practice 2,* Philadelphia, 1986, WB Saunders.
10. Miller JE: Parasites affecting goats in the Southeast, *Proceedings of Goat Production and Marketing Opportunities in the South,* 1995, Fort Valley, GA.
11. Uriarte J, Valderrabano J: Grazing management strategies for the control of parasitic diseases in intensive sheep production systems, *Vet Parasit* 37:342, 1990.
12. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
13. Herd RP, Zajac AM: Helminth parasites of the gastrointestinal tract. Nematode infections in cattle, sheep, goats, and swine. In Howard JL, Smith RA, editors: *Current veterinary therapy 4,* Philadelphia, 1998, WB Saunders.
14. Pugh DG, Navarre CB: Parasite control programs in sheep and goats, *Vet Clin North Am: Food Anim Pract,* 17(2):, 2001.
15. Wescott RB, Foreyt WJ: Epidemiology and control of trematodes in small ruminants, *Vet Clin North Am: Food Anim Pract* 2:373, 1986.
16. Miller DK, Craig TM: Use of anthelmintic combinations against multiple resistant *Haemonchus contortus* in Angora goats, *Small Rum Res* 19:281, 1996.
17. Hollis A et al: Preliminary investigation of anthelmintic efficacy against GI nematodes of goats and susceptibility of goat kids to gastrointestinal nematode infection, *Proceedings of the ARD Symposium,* 2000, Fort Valley, GA.
18. Greig A: Johne's disease in sheep and goats, *In Pract* 22(3):146, 2000.
19. Smith MC: Paratuberculosis in small ruminants, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
20. Stehman SM: Paratuberculosis in small ruminants, deer, and South American camelids, *Vet Clin North Am: Food Anim Pract* 12(2):441, 1996.
21. Clarke CJ et al: Comparison of the absorbed ELISA and agar gel immunodiffusion test with clinicopathologic findings in ovine clinical paratuberculosis, *Vet Rec* 139(29):618, 1996.
22. Thomas GW: Paratuberculosis in a large goat herd, *Vet Rec* 113(20):464, 1983.
23. Shulaw WP et al: Serodiagnosis of paratuberculosis in sheep by use of agar gel immunodiffusion, *Am J Vet Res* 54(1):13, 1993.
24. Pepin M, Marly J, Pardon P: *Corynebacterium pseudotuberculosis* infection in sheep and the complement fixation test for paratuberculosis, *Vet Rec* 120(10):236, 1987.

INTESTINAL OBSTRUCTION

Any cause of intestinal obstruction that occurs in other ruminants may occur in sheep and goats. Most of these diseases produce abdominal discomfort and occasionally abdominal distention. Diagnosis can be difficult because rectal palpation cannot be performed on small ruminants. Abdominal radiographs and ultrasonography may help differentiate these diseases, but exploratory surgery may be required to obtain a definitive diagnosis and select appropriate treatment.

Intussusception

Intussusception is more common in young animals, but it can occur in adults. It occurs when one segment of the intestine telescopes into an adjacent segment. Any portion of the intestine can intussuscept, but the ileum and ileocecal junction are the most common areas involved. When intussusception occurs, the lumen of the intestine narrows to the point of obstruction. The initiating cause is not always known.[1,2] It is associated with an intestinal mass in adults and enteritis in young animals.[1] *Oesophagostomum* infestations have been implicated as a cause in sheep.[1]

Clinical signs. The initial complaint is colic (manifested as kicking at the abdomen, repeated rising and lying down, and vocalization) followed by low-grade pain. True colic signs are variable in lambs and kids. In some cases, after the initial colic episode subsides, animals show no evidence of pain until the abdomen becomes distended. The time between the initial intussusception and abdominal distention depends on where the blockage occurs. Intussusception of the ileal area may take several days to cause bilaterally symmetric abdominal distention. Fecal output is scant, and what little there is may be dark or tarry, or may contain mucus. Dehydration becomes evident, hypochloremic metabolic alkalosis may develop and rumen chloride levels may increase with obstructions of the duodenum.

Diagnosis. Abdominocentesis may yield fluid compatible with a transudate (increased protein concentration and leukocyte numbers).[1] Radiography and ultrasonography reveal fluid-distended intestinal loops. Occasionally the intussusception itself can be visualized with ultrasonography or palpable through the abdominal wall. If the disease is not treated, intestinal rupture and peritonitis can occur.

Treatment. Surgical correction is required. If the intussusception is corrected early, the prognosis is good in the absence of peritonitis. Fluid support is needed to correct dehydration and metabolic abnormalities. Fluids should be administered IV until rumen function returns. Ringer's solution with added calcium (approximately 25 ml calcium borogluconate per liter) and potassium (10 to 20 mEq/L) is a good choice for fluid therapy.

Foreign Body Obstruction

Ingested foreign bodies or bezoars can obstruct portions of the intestines.[3,4] The signs are similar to those of obstruction caused by intussusception and depend on the part of the intestine that is blocked. In some cases the obstructing body can be seen with radiography or ultrasonography. Surgical removal is required for treatment.

Cecal Volvulus and Torsion of the Root of the Mesentery

Cecal volvulus and torsion of the root of the mesentery occur sporadically in sheep and goats.[1,3] Extreme abdominal pain, rapid abdominal distention, and circulatory collapse are typical signs. Immediate surgical correction and circulatory support are needed.

Intestinal Atresia

Atresia of the colon, rectum, and anus can all occur as congenital problems. The clinical sign of progressive abdominal distention usually is noted in the first week of life. Atresia of the anus can be diagnosed on physical examination, but atresia of the colon and rectum may require contrast radiography for a definitive diagnosis. Surgical establishment of anal patency can be performed for atresia ani. A permanent colostomy may be required for atresia of the colon and rectum. Atresia of the anus and rectum are considered heritable in cattle.[1] In the authors' experience, atresia ani is more common in sheep than in goats.

If surgical correction of atresia ani is attempted, the animal should be neutered or kept out of the breeding program because of the potential genetic basis for this condition. Occasionally a slight bulge in the skin may occur where the anus should be located, especially in male lambs. Ultrasonography can be used to locate a feces-filled rectum. For surgical correction, the clinician should locate the area where the anus should be, prepare it with sterile technique, and infiltrate it with a local anesthetic. The surgeon then makes a circumferential incision to remove the overlying skin covering the rectum. An alternative is to make an X-shaped incision into the rectum. Treated animals should be given mineral oil, DSS, or stool softeners as needed.

Intestinal Ileus

Ileus of the small intestine is a pseudo-obstruction that occurs when there is an absence of intestinal motility. The animal's failure to pass ingesta leads to signs similar to intussusception. The cause of ileus is usually unclear, but it

often occurs secondary to systemic diseases. The same elements that cause rumen stasis may potentially result in intestinal stasis and ileus. Symptomatic treatment with NSAIDs for pain and inflammation and fluids for dehydration is usually curative.[1] However, if signs persist, surgical exploration is indicated to rule out true obstructive diseases.

Peritonitis

Pathogenesis. Infection of the peritoneal lining of the abdominal cavity may lead to septic peritonitis. Common causes include uterine tears; rupture of the rumen or abomasum secondary to rumenitis, abomasitis, or abomasal ulcers; trocarization of the rumen for bloat; and rupture of the intestine secondary to obstruction.

Clinical signs. Signs depend on the severity of the condition. Abdominal discomfort and distention, dehydration, injected mucous membranes, depression, and death can all occur in cases of peritonitis. The presence of a fever is variable, both heart rate and respiration rate are usually elevated, and respiratory effort may be guarded. Animals may be febrile early, but have a normal to low body temperature as the condition progresses.

Diagnosis. Abdominal ultrasound can be useful in locating pockets of fluid for abdominocentesis, which usually yields fluid with increased protein concentration and leukocyte numbers. On occasion, intracellular bacteria are observed on cytologic examination. The presence of extracellular bacteria is not diagnostic because accidental enterocentesis can occur. Culture of abdominal fluid and subsequent antimicrobial sensitivity tests are indicated for the implementation of proper treatment. The causative organisms vary depending on the source of the bacteria. Rumen bacteria are typically gram-negative anaerobes, and *E. coli* and other enteric bacteria are common if the intestine is the source of infection. Exploratory surgery may be required to diagnose a gastrointestinal rupture. The CBC can be normal but often shows an inflammatory leukogram and, in severe cases, a degenerative left shift.

Treatment. Treatment includes the prescription of appropriate antimicrobial agents, the administration of NSAIDs for pain and endotoxemia, and fluid support for dehydration. The prognosis is guarded, especially if an intestinal rupture has occurred.

Rectal Prolapse

Pathogenesis, clinical signs, and diagnosis. Rectal prolapse is more common in sheep than in goats. This evagination of the rectal mucosa and rectal structures (and possibly the descending colon) is usually associated with excessive straining. Straining is seen in lambs with diarrhea caused by coccidiosis, *Salmonella,* or dietary imbalances, in ewes or ewe lambs with vaginal prolapse, in males with urolithiasis, and in animals grazing lush forage (particularly legumes such as alfalfa and clover). It also can occur secondary to chronic coughing, short tail docking, and the use of growth implants.[5-7] Rabies also can cause chronic straining and rectal prolapse.[5-9] Regardless of the cause, after the rectal mucosa becomes everted and exposed, irritation of the mucosa causes further straining, which exacerbates the problem. Venous drainage of the prolapse may be compromised, but the arterial supply usually remains intact and contributes to the swelling. Rectal prolapses are graded as Type I to IV, based on the portion of rectum and distal colon that is everted.[5] A description of these grades in shown in Table 4-4.

Treatment. Correction may be cost prohibitive for feedlot lambs, and immediate slaughter is recommended. In more valuable animals, very mild, early cases can be treated with frequent application of hemorrhoidal ointment designed for humans and manual replacement of the prolapsed mucosa into the anus. The authors try to avoid applying purse-string sutures in the anus because they tend to serve as a nidus and result in further straining. However, if less aggressive therapies do not relieve the problem in 24 hours, a purse-string suture may become necessary, particularly in Type I and II prolapse. In all cases and modes of treatment, restricting feed for 24 to 48 hours and administering mineral oil is recommended. Dusty feedstuffs (concentrates, pellets, hay) should be avoided because they may contribute to coughing, which exacerbates this condition. Adding molasses to feeds and lightly wetting hay may help reduce problems with dust.

Purse-string suture is easily performed. The prolapsed tissue and perineal area are washed with mild soap and lubricated with petroleum jelly or hemorrhoidal ointment before the prolapse is replaced.[5,9] After replacing the prolapsed mucosa, the clinician inserts a tubular object (syringe case, wooden dowel, gloved finger) into the rectum. He or she then places a purse-string suture of nonabsorbable suture material (3-5 nylon) in the skin around the anus, tightens it around the tubular object, and ties it off. The suture should be placed around the anus using a cutting needle, and entering and exiting at the 12 o'clock position. Tying the knot above the anus ensures that less fecal soiling of the suture will occur. The clinician should tie the suture in a bow knot to allow easy identification over the next few days and then remove the tubular object. The suture should be tight enough to prevent prolapse but loose enough to allow feces to pass. The clinician should regularly reevaluate the animal and if possible loosen the purse-string suture at 24-hour intervals until no tension exists. After a full day of no

TABLE 4-4

GRADES OF RECTAL PROLAPSE

TYPE	DESCRIPTION	COMMENTS
Type I	Small, circular amount of submucosal swelling protrudes through anus; probing reveals a pocket or fornix just inside anus	Good prognosis if there is no damage to mucosa; purse-string suture, iodine injection, submucosal resection
Type II	Slightly more circular submucosal and mucosal swelling, possibly containing retroperitoneal rectal tissue from anus; probing reveals a pocket just inside anus	Good prognosis if treated quickly and no mucosal damage; purse-string suture, iodine injection, submucosal resection, rectal amputation
Type III	Complete prolapse containing part of the retroperitoneal structures of the rectum and the descending colon; probing reveals a fornix just inside anus; the affected portion of the descending colon does not prolapse through the anus	If there is vascular injury to the descending colon, prognosis is guarded to poor; submucosal resection or rectal amputation are the methods of choice
Type IV	The descending colon appears as a tube, and has intussuscepted through the rectum and anus; unlike the previous types, in this case a probe or finger can be inserted into the prolapse through the anal sphincter for a distance of 5 to 10 cm	If there is vascular injury to the descending colon, prognosis is poor; abdominal exploration may be required to determine the extent of damage to the descending colon

From Hooper RN: General surgical techniques for small ruminants: Part II, *Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.

tension, the suture can be removed. If animals continue to strain, an epidural anesthetic can be administered. Petroleum jelly and hemorrhoid gels should be placed on the anus daily.[5,9]

The **injection of counterirritants** around the rectum (1 ml or less of Lugol's iodine) either alone or in conjunction with anal purse-string suturing is a quick and inexpensive treatment.[5,6,9] The clinician inserts an 18-gauge needle (5 cm) deeply into the skin around the anus at 12, 3, and 9 o'clock. An injection at the 6 o'clock position should be avoided because swelling around the urethra can result in obstruction.

For more severe cases, submucosal resection or rectal amputation of tissue may be necessary.[5,9] Rectal amputation can be performed with a prolapse ring or suture technique.[7] **Prolapse ring** usage is a salvage technique. The clinician inserts the prolapse ring into the rectum and places an elastrator band or suture around the area to be amputated to induce vascular compromise and necrosis of tissue. If a ligature is used, it should be tightened to allow purchase on the tube or ring. A fibrosis is induced just proximal to the band or suture, and mucosa subsequently grow across the areas.[5] Strictures, peritonitis, and abscesses are possible complications, but this technique may be useful as a field procedure.

Submucosal resection can be performed under epidural analgesia after the prolapse and the perineal area have been surgically prepared. The clinician places two spinal needles (9 to 10 cm) at 90-degree angles to each other 2 to 4 mm distal to the anal sphincter and through the entire prolapse.[5]

A circular incision is made 2 to 4 mm distal to the spinal needles through the mucosa and around the outside of the anus. Another circular incision is made just distal to the caudal extent of the prolapse into the point where the mucosa reflects on itself on the innerside of the prolapse. The clinician connects these two incisions with a longitudinal incision parallel to the prolapse and dissects the mucosa between the circumferential incisions.[5] The mucosal edges are then sutured with a simple interrupted pattern using a suitable absorbable suture material. After completely suturing the mucosal surfaces, the clinician removes the two spinal needles and places a purse-string suture in the anal sphincter. Placement of the suture and follow-up care are the same as described for the purse-string suture technique. Submucosal resection decreases the incidence of both peritonitis and stricture formation compared with other surgical techniques, but it is expensive.[5]

In all of these techniques, a caudal epidural anesthetic (2% lidocaine, 0.5 ml per 45 kg) is recommended to decrease straining and ease pain from the procedures.[6,7] A xylazine epidural (0.01 to 0.03 mg/kg as sufficient [QS] to 2 ml with 2% lidocaine) may give longer relief (approximately 4 to 6 hours) from straining than lidocaine. An alcohol epidural also may prevent straining for extended periods. Either isopropyl alcohol or ethanol can be used to demyelinate the motor and sensory nerves.[5] This type

of anesthesia can be permanent and therefore should be used only for animals intended for slaughter. Because of the potential for some loss of sciatic nerve function, the clinician should perform a test injection of a local anesthetic (2% lidocaine) before using alcohol. If the epidural appears effective and no ataxia or muscle weakness of the rear limbs occurs, the clinician can inject a mixture of equal parts of lidocaine and alcohol into the sites where the test epidural was performed. Possible problems with alcohol epidural anesthesia include injection site necrosis, sciatic nerve dysfunction, and the inability to void feces.[5]

Regardless of the type of epidural used, the clinician clips, washes, and dries the area before placing a small needle (20- to 21-gauge [2.6 cm]) in the most cranial yet moveable intracaudal vertebral space—usually C1 to C2 or C2 to C3. The needle is placed on the dorsal midline, with the needle 90 degrees to the skin and the hub moved slightly caudal, and then slowly advanced.

References

1. Guard C: Obstructive intestinal diseases. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
2. Mitchell WC: Intussusception in goats, *Agri-Practice,* p 1918, Dec 1983.
3. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
4. Sherman DM: Duodenal obstruction by a phytobezoar in a goat, *J Am Vet Med Assoc* 178:139, 1981.
5. Hooper RN: General surgical techniques for small ruminants: Part II, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
6. Pipkin AB: Rectal prolapse in ruminants and horses. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
7. Kimberling CV: *Jensen and Swift's diseases of sheep,* ed 3, Philadelphia, 1988, Lea & Febiger.
8. Welker B, Modransky P: Rectal prolapse in food animals. Part 1: cause and conservative management, *Comp Cont Ed Pract Vet* 13:1869, 1991.
9. Welker B, Modransky P: Rectal prolapse in food animals. Part II: surgical options, *Comp Cont Ed Pract Vet* 14:554, 1992.

DISEASES OF THE LIVER

Liver Abscesses

Liver abscesses usually occur as a result of chronic rumenitis in cattle, but they are rare in sheep and goats. They can occur in feedlot lambs and kids and other animals fed rations high in grain. In lambs and kids, septicemia or extension of an umbilical vein infection can cause liver abscesses.[1] In most cases, however, liver abscesses are an incidental finding. Weight loss, anorexia, depression, and decreased production (growth, milk) may occur. In adults, *Corynebacterium pseudotuberculosis* is the most common cause. *Actinomyces pyogenes* and *Fusobacterium necrophorum* also are cultured from abscesses.[1-3] Liver enzymes may or may not be elevated. Diagnostic ultrasonography of the liver may help detect abscesses, especially if they are numerous and widespread. However, no specific treatment or control measure is available. Many of the preventive protocols used for feeder cattle apply to the control of abscesses in sheep and goats. These include slowly introducing concentrates into the diet, offering long-stemmed hay free choice, and including rumen buffers (alkalinizing agents) and antimicrobial agents in the feed.

Pregnancy Toxemia/Fatty Liver Syndrome

Pathogenesis. Fatty liver occurs in conjunction with pregnancy toxemia in ewes and does during the last month of gestation. It is most common in thin or obese ewes or does with a single large fetus, twins, or triplets.[1] During late gestation, particularly in obese females, the abdominal space is filled with accumulated fat and an ever-expanding uterus. Because of the lack of rumen space, these females have difficulty consuming enough feedstuffs to satisfy energy requirements. In most management systems, late gestation occurs during the winter months, when less pasture is available and poorer-quality feedstuffs are offered. Energy requirements for ewes and does carrying twins or triplets is greatly increased during the final 2 months of gestation because 70% to 80% of fetal growth occurs during this time. Ewes with twins require 180% more energy, and those with triplets need 200% to 250% more dietary energy. Pregnancy toxemia also occurs in association with anorexia caused by other diseases (foot rot, OPP, CAE) or sudden stresses (feed or weather changes, predator attacks, hauling). Whatever the initiating cause, a period of anorexia and lack of sufficient energy intake result in a negative energy balance. These animals begin to mobilize body stores of fat and transport them to the liver. In the liver, fat is catabolized to glycerol and free fatty acids (FFAs). FFAs can be used in the citric acid cycle (Krebs cycle) as an energy source, but not in the direct formation of glucose. Anorexic animals have less ruminal substrate available for production of the glucose precursor propionic acid. However, oxaloacetate, which is an integral part of the citric acid cycle, is removed from the cycle and converted into glucose. Depletion of oxaloacetate inhibits the normal citric acid cycle's function, inhibiting the use of FFAs. As the pool of FFAs increases, they are converted to ketone bodies or repackaged into lipoproteins. Because ruminants are not efficient at transporting lipoproteins out of the liver and back to the adipose stores, the lipoproteins overwhelm the liver's ability to handle this massive buildup, resulting in a fatty liver. Because less substrate is available for glucose formation, more oxaloacetate is "cannibalized" from the citric acid cycle, further inhibiting the body's ability to use FFAs. This in turn causes the continued accumulation of ketone bodies. Hypoglycemia, hy-

perketonemia, and potentially uremia and death can occur.

Clinical signs. Animals suffering from fatty liver or pregnancy toxemia become anorexic and depressed, display altered behavior, and become recumbent. Some are constipated, grind their teeth, have a ketone smell to their breath, and suffer from dystocia. Neurologic signs include blindness, circling, incoordination, star-gazing, tremors, and convulsions.[4,5] Death can occur if the condition is left untreated. In the case of in-utero fetal death, maternal septicemia-endotoxemia and death are common sequelae.

Diagnosis. Diagnosis is based on clinical signs, the presence of multiple fetuses, and typical clinicopathologic findings. CBC results may be normal or show an eosinophilia, neutropenia, and lymphocytosis. These animals may or may not be hypoglycemic, but ketoacidosis, hypocalcemia, and hypokalemia are common.[2,4] Liver enzymes are usually within normal limits but occasionally may be increased. Azotemia, both from dehydration and secondary renal disease, is a common finding, and a fatal uremia may occur. Blood concentrations of ß-hydroxybutyric acid greater than 7 mmol/L are consistent with pregnancy toxemia. Urinalysis will be positive for both ketones and protein. Urine is collected from sheep by holding the nares and from does by frightening them and then allowing them a perceived escape when they stop, squat, and void. Although not commonly performed, liver biopsy can help determine the extent of fatty infiltration. This syndrome must be differentiated from hypocalcemia, hypomagnesemia, polioencephalomalacia, encephalitis, lead toxicity, and cerebral abscesses.

Treatment. Very early cases (before the animal exhibits recumbency) may be treated with oral or IV glucose. A balanced electrolyte solution with extra calcium (25 ml of a 23% calcium borogluconate per liter), potassium (10 to 20 mEq/L), and 5% dextrose is needed. In some cases, sodium bicarbonate is valuable in treating acidosis (see Appendix II). Energy intake must be increased, and propylene glycol can be administered (15 to 30 ml every 12 hours) as a glucose precursor. Rumen transfaunation and supplementation with vitamin B complex (including vitamin B_{12}, biotin, niacin, and thiamine) also are recommended. After females become recumbent, treatment must be very aggressive. Removal of the fetuses is crucial in these cases. Chemically inducing parturition (by administering 2.5 to 10 mg of prostaglandin $F_{2\alpha}$ or 0.75 μg/45 kg of cloprostenol in does and 15 to 20 mg of dexamethasone in ewes) and giving the ewe or doe medical support (fluids, B vitamins, glucose) while waiting is a useful protocol in some cases. Unfortunately, during the time before parturition, endotoxemia from dead fetuses further compromises the female. For this reason, the authors recommend immediate Cesarean section on depressed moribund animals (see Chapters 6 and 16). The owner should be forewarned of the poor prognosis for animals already in a moribund state. Fluid support during and after surgery is crucial.

Regardless of the therapeutic plan, the animal should be offered a palatable, energy-rich, highly digestible feedstuff. The keeper and clinician should take care to minimize the risk of a confounding disease during convalescence (e.g., lactic acidosis, polioencephalomalacia).

Prevention. Fatty liver and pregnancy toxemia can be prevented through proper nutrition. Maintaining animals in proper body condition throughout the year and making sure energy and protein levels are adequate in late gestation (see Chapter 2) are two key preventive measures.[2,4] The owner/manager should be taught to assess body condition in individual animals and should maintain emergency stores of feed in case of severe weather or natural disasters. The requirement for energy may be one and a half to two times maintenance for single fetuses and two to three times maintenance for multiple fetuses. Prevention of concurrent disease that may further increase energy demands or cause anorexia (e.g., intestinal parasitism, foot rot) is crucial. The keeper should take care to increase the grain portion of the diet slowly because anorexia from rumen upset can lead to this disease.[2] Ewes should be offered 0.5 to 1 kg of a cereal grain (corn, oats, barley, or a combination) every day during the final months of gestation; does can be offered ½ to 1 kg of grain. Keepers should maintain ewes and does at a body condition score of 2.5 to 3 (see Chapter 2) throughout gestation and evaluate the animals' energy every 2 to 4 weeks.

Ultrasonography can help identify females with multiple fetuses. These animals should be separated into groups and fed accordingly. Ultrasonographic determination of fetal numbers is best accomplished between days 45 and 90 after breeding with a 3.5 mHz transducer; a 5 mHz transducer produces better results between days 45 to 50. Either type of transducer may be of value and these windows of time may be expanded by the ability of the operator (see Chapter 6). Determination of fetal numbers may be enhanced by shearing the hair or fiber in front of the udder, applying a coupling substance to the skin, and viewing as much of the abdomen as possible, building a mental image of its structures and the number of fetuses while systematically moving from one side of the posterior abdomen to the other.

Keepers and clinicians should ensure that ewes are healthy and free of chronic diseases (e.g., OPP, CAE, foot rot, chronic parasitism) and that a good-quality trace mineral salt mixture is available free choice. The addition of lasalocid (0.5 to 1 mg/kg/day) or monensin (1 mg/kg/day) to the feed or mineral mixture enhances the formation of the glucose precursor propionic acid and

improves the efficiency of feed use. However, monensin should be used with caution because toxicity may occur; the agent should comprise no more than 30 ppm of the complete diet. The inclusion of niacin (1 g/head/day) in a feed supplement or mineral mixture helps prevent pregnancy toxemia. Supplementation with lasalocid, monensin, or niacin should begin 2 to 4 weeks before the females give birth.

Shearing in the last trimester also is recommended in ewes.[5] Many sheep producers routinely clip the wool around the vulva. If complete body shearing is performed, the incidence of fatty liver or pregnancy toxemia may be decreased. Sheared sheep require less energy to walk and graze. Sheared ewes also tend to shiver on cold days, exercising the enzyme systems that promote the more efficient use of FFAs as energy substrate. These ewes tend to seek shelter during cold weather, which may decrease lamb losses resulting from hypothermia. Obviously, if ewes are to be shorn, keepers should make adequate shelter available.

Keepers should avoid hauling or moving females during late gestation. Proper predator control measures should be maintained. Good hoof care programs should be in place on farms or ranches where grazing is the predominant form of nutrient intake. Sheep and goats should have their teeth checked to ensure good dentition before the breeding season. Animals with poor teeth should be culled.

Measuring serum β-hydroxybutyric acid concentrations is useful in assessing energy status in ewes. Values of 0.8 to 1.6 mmol/L suggest a negative energy balance. Keepers should take steps to correct the problem by feeding better-quality, more digestible feedstuffs.

White Liver Disease

White liver disease is a form of fatty liver disease reported only in Angora and Angora-cross goats and sheep. It is associated with cobalt deficiency.

Pathogenesis. Cobalt is needed by rumen microflora to produce cyanocobalamin, or vitamin B_{12}, which is a coenzyme for methylmalonyl-CoA mutase. In turn, this enzyme is needed to convert propionate to glucose through the Krebs cycle. Cobalt deficiency leads to the accumulation of methylmalonyl-CoA, or methylmalonic acid, which is converted to branched chain fatty acids that accumulate in the liver.[2,6] High-grain diets that are fermented to propionate coupled with deficient or marginal cobalt intake may predispose to this condition.[2,6] White liver disease has not been reported in the United States, but ill thrift from cobalt deficiency has been observed. It is therefore possible that the disease goes unrecognized.[1]

Clinical signs. Signs are most commonly seen in young animals, and include ill thrift, anorexia, and diarrhea; sheep may exhibit photosensitization. Clinicopathologic findings include a macrocytic, normochromic anemia and hypoproteinemia.[1,2]

Diagnosis. Abnormal serum or liver concentrations of vitamin B_{12} or liver cobalt are the basis of diagnosis. Liver cobalt concentrations on a dry matter basis of 0.08 ± 0.02 ppm were reported in goats with white liver disease, compared with 0.53 ± 0.11 ppm in controls.[6]

Treatment and prevention. Sheep can be treated with oral cobalt (1 mg/head/day) or vitamin B_{12} injections. The condition can be prevented by including cobalt in the ration by feeding a good-quality trace mineral salt.[2]

Liver Flukes

Both *Fasciola hepatica* and *Fascioloides magna* can infest sheep and goats. The disease occurs along the Gulf Coast and in the Pacific Northwest and Great Lakes areas.

Pathogenesis. The life cycles of *F. hepatica* and *F. magna* are similar in that each requires an aquatic snail as an intermediate host. Fluke eggs that are passed in animal stool hatch in water to form miracidia, which penetrate the intermediate host. The miracidia develop through several intermediate hosts to form cercariae, which emerge from the intermediate hosts to encyst as infective metacercariae on forage. Sheep or goats accidentally ingest the metacercariae, which then encyst in the small intestine. They can migrate into the liver in approximately 6 weeks and may begin to lay eggs within 3 months after infection. *F. hepatica* is capable of laying eggs in sheep for several years. The flukes also can migrate into the bile ducts or through hepatic tissue, leaving large anaerobic tracts and producing acute or chronic disease.[7] Sheep and goats are definitive hosts for *F. hepatica,* whereas only some species of deer and elk are definitive hosts for *F. magna. F. magna,* unlike *F. hepatica,* never matures and continues to migrate, causing severe damage and death.[7,8] *F. magna* does not complete its life cycle in sheep or goats. The most serious complication of acute liver fluke infestation is black disease *(Clostridium novyi),* which presents as sudden death.

Clinical signs. *F. hepatica* infestation usually causes acute disease in sheep and goats but can present as a chronic condition. Chronic disease is the result of the mature flukes in the bile ducts and is manifested in depressed growth and milk production. Acute disease occurs when large numbers of immature flukes migrate at once, particularly in animals with limited immunity to flukes. Signs include anorexia, depression, weakness, dyspnea, anemia, ascites, colic-like signs, dry feces, and

sudden death. The clinical signs are identical to those of nematode infestations (i.e., chronic weight loss, ill thrift, diarrhea, anemia, hypoproteinemia). Similar but more severe signs occur with *F. magna* infection, which is usually fatal.[2,5,8]

Diagnosis. Antemortem diagnosis of fluke infestation can be difficult. Finding eggs in feces is diagnostic for *F. hepatica.* Eggs are only produced by adults and not in great numbers, so a negative fecal test cannot preclude acute or chronic fascioliasis. Fluke eggs do not float in routine fecal flotation methods used for nematode diagnosis; a sedimentation technique should be used for suspected fluke infestations. To perform a sedimentation test, the clinician mixes 2 to 3 g of feces with 200 ml of tap water and strains the mixture through a tea strainer into a beaker. The sediment can be examined 15 minutes later under a dissecting microscope. Eggs are light yellow to golden and have an operculum at one end (see Figure 4-10). *F. magna* does not mature, so eggs are not produced and fecal examination is of no value. Most fluke infestations are discovered by finding the flukes at necropsy or slaughter. An ELISA test may be available in the future.[2,5,8] CBCs of affected animals may indicate eosinophilia and anemia. Increased liver enzymes and hypoalbuminemia also are occasional findings.

Treatment. Because antemortem diagnosis is difficult, the clinician should institute fluke treatment after ruling out other differential diagnoses if the possibility of fascioliasis exists. If fascioliasis is diagnosed at necropsy, the remaining animals in the herd should be treated. Because flukicides available in the United States are highly effective only against mature flukes, the timing of treatment is important. In the southern portions of North America the snails are ingested in the spring and the flukes migrate in the summer and mature in the fall. In cooler, northern climates, snails may remain active during summer, so flukes can mature in the fall and into the winter. Clinicians should begin treatment in the southern United States in the late summer or early fall. A single treatment in late winter or early spring is commonly used in the northern climates of North America. Albendazole (15-20 mg/kg orally) and clorsulon (7 mg/kg orally, 2 mg/kg SC) are very effective against adult *F. hepatica.*[2,9,10] Clorsulon has no efficacy against nematode parasites but is highly efficacious against both adult and late-stage immature flukes. Albendazole (15 mg/kg orally) is somewhat useful in controlling *F. magna* at 8 weeks after infestation, and clorsulon is effective only at very high dosages.[11-13] Unfortunately, neither agent can kill 100% of *F. magna,* and only a few remaining flukes can be fatal.

Prevention. Control of fluke infestations is difficult, although timely treatment of animals can decrease infections in successive years. Decreasing exposure is the key to control. Eliminating the snail is impractical, but fencing off low-lying areas may prevent ingestion. Depending on local fluke life cycles, keepers should avoid grazing animals on areas with high fluke populations during peak infection times. Areas where water stands or flows over grazing pastures, streams, and irrigation ditches (particularly those with clay soil) are high-risk zones.

Cysticercosis

Cysticercus tenuicollis is the larval stage of the dog tapeworm *Taenia hydatigena,* of which sheep and goats are intermediate hosts. The larval stage migrates through the liver, then attaches to the liver or other abdominal organs and causes black, winding tracts and cysts in the liver. Acute disease occurs only with large numbers of cysticerci and is characterized by depression and weakness resulting from liver damage. The chronic cystic stage is usually asymptomatic. No treatment is available and control is problematic because it requires treating infestation in dogs and preventing contact with dogs.[1,2,5]

Copper Toxicosis

Pathogenesis. Copper (Cu) toxicosis is more common in sheep than in goats. Goats appear closer to cattle than sheep in their ability to store and handle Cu and resist toxicosis. Toxicity results from chronic accumulation in the liver from the ingestion of excess Cu in relation to molybdenum (Mo) or sulfate in the diet. In sheep, a Cu-to-Mo ratio greater than 10:1 leads to the accumulation of excess Cu. The most common sources of excess Cu in sheep and goats are trace mineral mixtures and feeds formulated for cattle or horses. Clinical signs are often absent during the chronic accumulation phase. Acute disease is seen when Cu is suddenly released from the liver in large amounts. Stress usually precipitates this acute phase. Acute release and subsequent high blood Cu concentrations cause an acute hemolytic crisis, resulting in anemia, hemoglobinuria, and acute renal failure. Existing hepatic disease (such as that caused by liver flukes) may predispose animals to this condition. Some breeds seem to be prone to Cu absorption and storage problems (Merino sheep), whereas others tend to be more resistant and prone to deficiency (pygmy goats) (see Chapters 2 and 3).

Clinical signs. Anorexia, depression, diarrhea, and weakness are all signs of Cu toxicity. Many affected animals are found dead with hemolysis and icterus. Signs of abdominal pain and diarrhea are sometimes present. Port wine–colored urine is evidence of hemoglobinuria. Hemoglobinemia produces icterus of the mucosal membranes and fever.

Diagnosis. On clinicopathologic examination, anemia, hemoglobinemia, hyperbilirubinemia, increased liver enzymes, and azotemia are present. Urinalysis reveals hemoglobinuria and isosthenuria. The combination of azotemia and isosthenuria indicates acute renal failure. Definitive diagnosis of acute disease requires measurement of Cu concentrations in serum. Normal blood Cu concentrations are approximately 50 to 200 μg/dl in sheep and goats.[2,5,14] These concentrations increase 10- to 20-fold with an acute hemolytic crisis.[5] On necropsy, kidney Cu concentrations are the most diagnostic because liver concentrations may be normal from release into the bloodstream. Generally kidney concentrations greater than 100 ppm and liver concentrations greater than 350 ppm on a dry matter basis are diagnostic.[2,5] If tissue copper is reported in wet weight, the conversion to dry tissue weight can be estimated by multiplying the tissue concentration by a factor of 3.5

Treatment. Treatment of acutely affected animals is often futile. It consists of supportive therapy for the acute renal failure and anemia and attempts to lower liver Cu stores. Fluid therapy for the acute renal failure (see Appendix II) is of therapeutic value, and a blood transfusion may be needed if the PCV drops precipitously. Ammonium tetrathiomolybdate (1.7 mg/kg IV or 3.4 mg/kg SC on alternate days for three treatments) is the most economical agent for treatment for acute cases. In valuable animals, D-penicillamine (26 to 50 mg/kg BID or 52 mg/kg SID PO for 6 days) increases urinary Cu excretion. Trientine is used in human beings, but has shown variable results in sheep.[15] Treatment of the remainder of the flock should include the administration of ammonium molybdate (50 to 500 mg/head/day PO) and sodium thiosulfate (300 to 1000 mg/head/day PO) for 3 weeks.[15] Stress should be minimized, so keepers and clinicians should delay routine maintenance procedures such as deworming and hoof trimming until after treatment. The offending source of Cu should be eliminated.

Prevention. Avoiding high dietary Cu (more than 10 ppm), a high Cu-to-Mo ratio (greater than 10:1) in the feed, Cu-containing foot baths, and other sources of Cu is crucial. Including supplemental Mo in the diet to lower the Cu-to-Mo ratio to 6:1 to 8:1 is beneficial. This requires 2 to 6 ppm of Mo in many instances. Often too much emphasis is placed on the trace mineral component of the diet. The clinician should be aware that even if no Cu is added to the trace mineral mixture and the element does not appear on the product label, the mineral mixture may still contain Cu. Many components of mineral mixes are contaminated with Cu (zinc sulfate may contain 400 ppm of Cu, dicalcium phosphate may contain more than 30 ppm of Cu). Therefore the clinician needs to perform a dietary analysis to find and correct the problem.

Toxic Hepatitis

Pathogenesis. The liver is vulnerable to toxic insult because one of its major functions is detoxification. The most common plants that are gastrointestinal and liver toxins are shown in Table 4-5. Clinical signs depend on the cause. Acute, severe toxicity is more common with chemical toxicosis, whereas plant toxins usually cause chronic disease. A thorough history is important and in many cases inspection of the animals' environment is required.

Clinical signs. The clinical signs of toxic hepatitis can be vague. Animals may only show anorexia and depression. Icterus is more common with hemolytic diseases and is not always seen with liver disease. Photosensitivity is a common clinical feature in ruminants and hepatoencephalopathy also can occur.

Diagnosis. Clinicopathologic data are more helpful in diagnosing acute toxicity. Serum AST and LDH levels can increase with hepatocellular necrosis but are not liver-specific, so muscle injury and disease must be ruled out. These enzymes also increase if serum is not separated from a blood clot in a timely fashion.[1] Increased levels of alkaline phosphatase (AP) and GGT indicate biliary stasis. AP also is not liver-specific, but increased serum levels of GGT are very specific for liver disease. GGT also increases in some hepatocellular diseases, so testing for its normal concentrations is important.[15] Unfortunately, all of these enzymes can be normal with liver disease, especially if it is chronic. Hyperbilirubinemia, hypoglycemia, low blood urea nitrogen (BUN), and hypoalbuminemia are not always evident as classically taught. If hepatoencephalopathy is suspected, blood ammonia concentrations may be elevated. Blood ammonia analysis may be impractical in the field because the blood should be kept on ice, and the test should be performed within 30 minutes of collection. To enhance the accuracy of blood ammonia analysis, the clinician should collect blood from a normal control animal for comparison. Ammonia concentrations three times those of the control animal are diagnostic.[16] Liver biopsy remains the most valuable tool in diagnosing liver disease. Although clotting dysfunction may occur in liver disease, it is an uncommon complication in ruminants and should not discourage the clinician from performing a liver biopsy.

Treatment. If the intoxication is caught in the acute stage, activated charcoal (500 g per adult animal) can be given. Supportive care, especially fluid support with dextrose solutions, is the mainstay of therapy. Low-protein diets may suppress ammonia production temporarily, but they can be detrimental over time depending on the production status of the animal. If photosensitization occurs, animals should be housed indoors if possible, and broad-

TABLE 4-5

PLANTS CAUSING GASTROINTESTINAL OR HEPATIC DISEASE

PLANT	COMMENTS	SIGNS
Cocklebur	Erect annual herbage in sandy soils, flood plains, and overgrazed pastures; seeds are toxic	Within hours to days of ingestion—anorexia, vomiting, colic, dyspnea, gastroenteritis, chronic hepatitis, hepatic damage, death
Senico-groundsel, Crotalaria, heliotropism, amsinckia (fiddleneck), echium	Pyrrolizidine alkaloid; excreted in milk and urine and can cross placenta; young more susceptible	Dullness, weakness, weight loss, icterus, fibrosis, hepatocytomegaly, bile duct proliferation, photosensitivity; subcutaneous edema, diarrhea
Lantana	Found in sandy, tropical areas; berries, leaves, and hay are toxic	Chronic toxicity—slow hepatic failure; icterus, photosensitization, weakness, bloody diarrhea, cholestasis, hepatic toxicity
Sneezeweed, bitterweed, rubberweed	Grows in overgrazed pastures; all parts of plant are toxic	Acute toxicity—gastrointestinal upset, depression, serous nasal discharge, salivation, bloat; chronic toxicity—vomiting, bittermilk lesions, hepatic and renal congestion, gastric edema, aspiration pneumonia; pulmonary edema
Cabbage, kale, rape, mustard, wild mushroom	Remove from diet; add iodine to diet (for goiter)	Gastroenteritis, hepatic necrosis, photosensitization, goiter, hemolysis
Horsebrush	Stop grazing, keep animals indoors	Bighead, itching, uneasiness, inflamed eyes, blindness, serum discharge from scabs; degenerative changes in liver and elevated liver enzymes
Clover (crimson, red, subterranean burclover)		Photosensitization
St. John's wort	Perennial herb; grows along roadsides and in overgrazed fields; remove from diet and keep animals in shade	Increased respiration, diarrhea, pruritus, dermatitis, diarrhea, death

spectrum (systemic or topical) antibiotics may be necessary to control secondary bacterial dermatitis. Corticosteroids (dexamethasone 0.1 to 1 mg/kg IV or IM) may be indicated in early cases of photosensitization to decrease inflammation. Neurologic signs can be controlled with phenobarbital (initial dose: 10 to 20 mg/kg IV diluted in saline and administered over 30 minutes; subsequent doses: 1 to 9 mg/kg IV diluted in saline, as needed up to TID). Diazepam (Valium) is contraindicated in hepatoencephalopathy because it may worsen signs.[17]

Miscellaneous Liver Diseases

Congenital hyperbilirubinemia, or black liver disease, occurs in mutant Corriedale sheep (Dublin-Johnson syndrome).[1] This is a genetically recessive condition. It is characterized by an abnormality in the excretion of conjugated bilirubin and phylloerythrin and is often seen in animals consuming green forage. Clinical signs include anorexia, photodermatitis, and icterus. Liver biopsy of affected animals reveals dark to black granules in otherwise normal hepatocytes. The syndrome first manifests itself in lambs around 5 months of age.[18]

A similar condition occurs in Southdown lambs around 6 months of age (Gilbert's syndrome). This too is a recessive condition that causes decreased hepatic uptake of phylloerythrin and bilirubin, with concurrent renal failure.[18] Signs include icterus, photodermatitis, and ulceration around the ears and mouth. A liver biopsy reveals normal hepatic tissue. In both of these conditions, animals should be kept out of sunlight and fed minimal

TABLE 4-5

PLANTS CAUSING GASTROINTESTINAL OR HEPATIC DISEASE—cont'd

PLANT	COMMENTS	SIGNS
Blue-green algae	Toxic after a bloom	Vomiting, diarrhea, liver failure, photosensitization; necropsy findings include swollen bloody liver, edema around gallbladder, centrolumbar apoptosis, and necrosis
Pokeweed		Vomiting, cramps, diarrhea, weakness, dyspnea, prostration, tremors, convulsions
Gossypol (cottonseed)	Toxicity seen in younger pre-ruminants	Poor performance, convulsions, cardiac toxicity
Rhubarb	Contains oxalic acid	Gastrointestinal toxicity
Oak	Acorns and oak buds are most toxic	Abdominal pain, pseudomembranes in gastrointestinal tract, bloody diarrhea, depression, renal toxicity
Castor bean	Beans most toxic	Gastrointestinal irritation, bloody diarrhea, central nervous system disturbances
Mistletoe	Berries not toxic	Nausea, diarrhea
Others:		
English ivy		
Sesbania		
Narcissus		
Elderberry		
Spurge		
Buckwheat		
St. Anne's lace		
Milkweed		
Parsley, giant hogweed		

amounts of green forage. Obviously, affected animals should be neutered or culled.

Various tumors of the liver, including fibrosarcoma, lymphosarcoma, and cholangiocellular carcinoma, have been reported.[17,18] The use of ultrasonography and ultrasound-guided liver biopsy may aid in diagnosis.

References

1. Fetcher A: Liver diseases of sheep and goats, *Vet Clin North Am: Large Anim Pract* 5:525, 1983.
2. Smith MC, Sherman DM: *Goat medicine,* Philadelphia, 1994, Lea & Febiger.
3. Santa Rosa J et al: A retrospective study of hepatic abscesses in goats: pathological and microbiological findings, *Br Vet J* 145(1):73, 1989.
4. Parson EG, Maas J: Hepatic lipidosis. In Smith BP, editor: *Large animal internal medicine,* ed 2, St Louis, 1996, Mosby.
5. Kimberling CV: *Jensen and Swift's diseases of sheep,* ed 3, Philadelphia, 1988, Lea & Febiger.
6. Johnson EH et al: Hepatic lipidosis associated with cobalt deficiency in Omani goats, *Vet Res Commun* 23(4):215, 1999.
7. Craig TM: Epidemiology of internal parasites: effects of climate and host reproductive cycle on parasite survival, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.
8. Wescott RB, Foreyt WJ: Epidemiology and control of trematodes in small ruminants, *Vet Clin North Am: Food Anim Pract* 2:373, 1986.
9. Foreyt WJ: Efficacy and safety of albendazole against experimentally induced *Fasciola hepatica* infections in goats, *Vet Parasit* 26(1-2):261, 1988.
10. Rehbein S, Visser S: Efficacy of an injectable ivermectin/clorsulon combination against *Fasciola hepatica* in sheep, *Vet Rec* 145(16):468, 1999.

11. Foreyt WJ, Foreyt KM: Albendazole treatment of experimentally induced *Fascioloides magna* infection in goats, *Agri-Practice,* p 1441, Sept 1980.
12. Conboy GA, Stromberg BE, Schlotthauer JC: Efficacy of clorsulon against *Fascioloides magna* infection in sheep, *J Am Vet Med Assoc* 192(7):910, 1988.
13. Foreyt WJ: Evaluation of clorsulon against *Fascioloides magna* in cattle and sheep, *Am J Vet Res* 49(7):1004, 1988.
14. Kaneko JJ: *Clinical biochemistry of domestic animals,* ed 4, San Diego, CA, 1989, Academic Press.
15. Plumlee KH: Metals and other inorganic compounds. In Smith BP, editor: *Large animal internal medicine,* St Louis, 1996, Mosby.
16. Roussel AJ, Whitney MS, Cole DJ: Interpreting a bovine serum chemistry profile: Part I, *Vet Med,* 92:553, 1997.
17. Divers TJ: Therapy of liver failure. In Smith BP, editor: *Large animal internal medicine,* St Louis, 1996, Mosby.
18. Ogilvie TH: *Large animal internal medicine*, Baltimore, 1998, Williams & Wilkins.

PATHOLOGY OF THE UMBILICUS

Umbilical Hernia

The umbilicus is an opening in the ventral abdominal wall that allows passage of the umbilical vessels and allantoic stalks. This opening should close within a few day of birth. The failure of this opening to close properly is termed *umbilical hernia.* The hernial sac has an inner peritoneal layer and an outer layer of skin. These hernias are probably of genetic origin but may occur as sequelae to umbilical remnant infection. The opening in the abdominal wall is perceived as a ring on palpation. If the clinician can insert more than one finger into the hernial ring or if the hernia persists for more than 3 to 4 weeks, surgical intervention is indicated.

Penning. Clamps or rubber bands may be of value for closing small hernias (those less than 4 cm in diameter). The clinician should either lightly sedate the animal or infiltrate the skin around the hernia with a local anesthetic (2% lidocaine). The animal should be placed on its back and held by a technician-helper. Any viscera prolapsing into the hernial sac should be replaced into the abdomen. The clinician then inserts two metal pins (baby diaper pins can be used) through the skin and on opposite sides of the hernial ring, just on the edge of the linea alba. The pins should be placed deep enough to sit next to the abdominal wall. Slight tension is placed on the skin in the center of the umbilical sac, pulling it away from the abdomen. When the clinician is confident that all viscera have been cleared from the hernial sac, he or she places an elastrator band between the pins and the abdominal wall. This results in ischemic necrosis of the skin. The skin will slough and the abdominal defect will heal in 7 to 14 days. Lambs should be given tetanus prophylaxis. This procedure and other clamping techniques are useful in females and some males. However, urine scalding of the skin may occur in some males. Clinicians should closely monitor animals that have undergone clamping.

Surgical resection. In cases in which the hernial ring is larger than 5 cm, surgical intervention should be carried out. Animals can be sedated and then infiltrated with a local anesthetic or placed under general anesthesia. The area around the hernia is clipped and surgically prepared. The clinician opens the hernial sac and introduces a finger into the abdomen to ensure that no viscera have adhered to the inner lining of the ring and that no enlarged or infected umbilical remnants are present. He or she then carefully excises the ring and closes the defect in the abdominal wall. This closure can be made by simply opposing the abdominal wall with a horizontal mattress pattern stitch (absorbable suture). An alternate closure of the abdominal wall is to suture the peritoneal lining in a separate pattern and close the abdominal wall defect so one side of the defect is pulled to overlap the other side. The upper free edge is sutured to the opposite wall with a near-far-far-near pattern. The authors choose not to employ surgical techniques that slow this procedure. The subcutaneous tissue can be closed with simple interrupted pattern using absorbable suture and the skin should be closed with whatever pattern the clinician prefers. Animals should be given tetanus prophylaxis and antibiotics. They should be closely monitored for signs of sepsis and surgical failure. Exercise should be limited for 7 to 14 days after surgery.

Umbilical Infections

Infections of the umbilical arteries (omphaloarteritis) and veins (omphalophlebitis) and urachal disease can occur because of failure or partial failure of passive transfer of colostral antibodies and subsequent sepsis. Contamination of the umbilicus, retracting of these structures after stretching and breaking, and chemical damage (from strong tincture of iodine) to the amniotic remnants are other possible causes.[1,2,3] Dipping the umbilicus with iodine or iodine-chloriodine substances is a common practice. Aggressive use of these chemicals may precipitate serious inflammation of the cord. Excessive torsion of the umbilical cord, distention of the proximal urachus, and some genetic factors may all be associated with patent urachus, which also may occur as a sequela to omphaloarteritis or omphalophlebitis.

Clinical signs and diagnosis. The clinical signs include umbilical swelling, pain, and occasionally drainage or discharge of the umbilical stump. Palpation and transabdominal ultrasonographic evaluation reveal an enlarged cord-like structure ascending from the umbilicus cranially (umbilical vein) or caudally (urachus or um-

bilical artery). Ultrasonographic evaluation may indicate an abscess or thickened tissue. Patent urachus is associated with dermatitis, urine scalding of the ventral abdomen, and urine dribbling. If the urachus becomes infected it may leak urine intraperitoneally or subcutaneously. Both of these developments may be identified with abdominal palpation, ballottement, ultrasonographic evaluation, and, when indicated, paracentesis.[1] The CBC may indicate neutrophilia. Blood culture is indicated if sepsis occurs simultaneously. Occasionally infection of the internal structures may occur with no outward umbilical swelling. Deep abdominal palpation and/or the use of real-time ultrasound are necessary to attain a diagnosis. Animals with umbilical infections also may have signs of septicemia, anorexia, depression, joint distention, and fever.

Treatment. If a patent urachus occurs without inflammation of the associated tissues, it can be cauterized daily with iodine or silver nitrate. However, if it remains patent for more than 5 days, it should be surgically closed. The animal should be placed under general anesthesia (see Chapter 16). The area around the umbilicus should be clipped and surgically prepared, and the animal should be placed on a broad-spectrum antimicrobial agent 2 to 4 hours before surgery. The clinician opens the abdomen lateral to the umbilicus and digitally explores the adjacent area for adhesion formation. The urachus should be identified and followed to the urinary bladder. After this, the clinician should amputate the urachal attachment to the bladder and close the bladder with a double-layered inverting pattern (Cushing). The abdominal wall, subcutaneous tissue, and skin are closed as described for umbilical hernia repair.

On occasion some cases of omphalophlebitis-omphaloarteritis can be treated medically. Prolonged antibiotic therapy with a broad-spectrum antimicrobial agent (ceftiofur 2.2 mg/kg SID or oxytetracycline 20 mg/kg SC every 72 hours) may be attempted. However, if medical therapy is ineffective, the infected umbilical remnants should be marsupialized or excised. The authors prefer more aggressive, surgical removal of the umbilical remnants. As with urachal surgery, the abdomen should be opened lateral to the umbilicus. Depending on the severity of infection and the amount of tissue involved, the clinician may need to perform extensive dissection of necrotic tissue and possibly intestinal resection.[3]

If the infection of the umbilical vein extends to and involves the liver, marsupialization of the umbilical vein is an effective method of therapy.[2,3] The clinician can pull the vein to the most cranial portion of the abdominal incision and suture it to the muscle layers and skin before closing the abdomen as described for umbilical hernia repair. However, a preferable method is to close the abdominal wall, pull the transected umbilical vein through, and suture it to a separate stab incision. This may help minimize the incidence of abdominal wall herniation. Only monofilament, absorbable, non-gut suture material should be used.[3] The venous stump should be flushed daily with antiseptic solution (1% chlorhexidine, 0.1% povidone iodine), and the animal should be maintained on antibiotics for more than 14 days. The venous stump usually closes within a month.[3]

Prevention. Umbilical infections can be prevented or drastically reduced by ensuring adequate intake of good-quality colostrum. Lambs and kids also should be only minimally stressed (particularly during the first 2 to 3 days of life) to enhance colostral absorption. In some management scenarios, proper dipping of the navel with non-caustic materials also helps reduce the incidence of this disease.

References

1. Lofstedt J: Neonatal conditions, with emphasis on equine neonate. In Ogilvie TM, editor: *Large animal internal medicine,* Baltimore, 1998, Williams & Wilkins.
2. Rings DM: Umbilical hernia, umbilical abscess, and auricle fistula, *Vet Clin North Am: Food Anim Pract* 11:137, 1995.
3. Hooper RN: General surgical techniques for small ruminants: Part II, *Proceedings of the Small Ruminants for the Mixed Animal Practitioner, Western Veterinary Conference,* 1998, Las Vegas, NV.